TRANSACTIONS

OF THE

TWENTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

339367
2. 7. 37.

HELD AT
MEMPHIS, TENN.,
DECEMBER 11, 12, 13, 1916

The Twenty-Second Annual Meeting of the American Academy of Ophthalmology and Oto-Laryngology will be held at Pittsburgh, Pa., October 29, 30, 31, 1917.

RE
1
A44
v.21

Officers for 1917

Dr. W. L. Dayton.....President
Lincoln, Neb.

Dr. Edward Stieren.....First Vice President
Pittsburgh, Pa.

Dr. Alexander C. Magruder.....Second Vice President
Colorado Springs, Colo.

Dr. John Green, Jr.....Third Vice President
St. Louis, Mo.

Dr. Secord H. Large.....Treasurer
Cleveland, Ohio.

Dr. Lee Masten Francis.....Secretary
Buffalo, N. Y.

COUNCIL

Ex Officio

Dr. W. L. Dayton.....Lincoln, Neb.
Dr. Edward Stieren.....Pittsburgh, Pa.
Dr. S. H. Large.....Cleveland, Ohio

Dr. John Edwin Brown.....Columbus, Ohio
Dr. Joseph C. Beck.....Chicago, Ill.
Dr. Walter B. Lancaster.....Boston, Mass.
Dr. Richmond McKinney.....Memphis, Tenn.
Dr. J. R. Newcomb.....Indianapolis, Ind.
Dr. J. M. Patton.....Omaha, Neb.

Officers for 1916

Dr. J. E. Brown.....President
Columbus, Ohio

Dr. T. E. Carmody.....First Vice President
Denver, Colo.

Dr. F. P. Calhoun.....Second Vice President
Atlanta, Ga

Dr. Kate Baldwin.....Third Vice President
Philadelphia, Pa.

Dr. Secord H. Large.....Treasurer
Cleveland, Ohio

Dr. Lee M. Francis.....Secretary
Buffalo, N. Y.

MEMBERS OF COUNCIL

Dr. J. E. Brown.....Columbus, Ohio
Dr. T. E. Carmody.....Denver, Colo.
Dr. S. H. Large.....Cleveland, Ohio
Dr. Joseph C. Beck.....Chicago, Ill.
Dr. J. M. Ray.....Louisville, Ky.
Dr. H. B. Young.....Burlington, Iowa
Dr. T. W. Moore.....Huntington, W. Va.
Dr. Walter B. Lancaster.....Boston, Mass.
Dr. Richmond McKinney.....Memphis, Tenn.

PRESIDENTS OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

ALT, ADOLF, St. Louis, Mo.....1896
ALT, ADOLF, St. Louis, Mo.....1897
FRYER, B. E., Kansas City, Mo.....1898
COLBURN, J. E., Chicago, Ill.....1899
SCHEPPEGRELL, W., New Orleans, La.....1900
GOLDSTEIN, M. A., St. Louis, Mo.....1901
HOLMES, CHRISTIAN R., Cincinnati, Ohio.....1902
BALLENGER, WM. L., Chicago, Ill.....1903
JACKSON, EDWARD, Denver, Colo.....1904
LOEB, HANAU W., St. Louis, Mo.....1905
WOOD, CASEY A., Chicago, Ill.....1906
STUCKY, J. A., Lexington, Ky.....1907
VAIL, DERRICK T., Cincinnati, Ohio.....1908
STEIN, OTTO J., Chicago, Ill.....1909
REBER, WENDELL, Philadelphia, Pa.....1910
KYLE, J. J., Indianapolis, Ind.....1911
SUKER, G. F., Chicago, Ill.....1912
MURPHY, JOHN W., Cincinnati, Ohio.....1913
RAY, J. M., Louisville, Ky.....1914
BECK, JOSEPH C., Chicago, Ill.....1915
BROWN, JOHN E., Columbus, Ohio.....1916
DAYTON, W. L., Lincoln, Neb.....1917

OPHTHALMOLOGY

TABLE OF CONTENTS

PAGE

President's Address.
JOHN E. BROWN, M.D. 1

The Internal Secretory System and the Eye.
ROBERT SCOTT LAMB, M.D. 5

Ring Scotoma in Its Relation to Choroidal and Optic Nerve Disease.
WENDELL REBER, M.D. 13

"Renal Choked Disk."
WILLIAM F. HARDY, M.D. 25

Permanent Occlusion of the Superior Branches of the Central Artery, Probably Due to Thrombosis of the Central Vein of the Retina.
GEORGE W. MACKENZIE, M.D. 39

Tuberculosis of the Retinal Vessels.
FRANK R. SPENCER, M.D. 60

Some Phases of the Diagnostic and Therapeutic Uses of Tuberculin in Uveitis.
HIRAM WOODS, M.D. 75

Visual Fields in Pellagra (A Preliminary Report).
F. PHINIZY CALHOUN, M.D. 85

Campimeter Versus Arc Perimeter.
LUTHER C. PETER, M.D. 94

The Excision of the Tarsus in Trachoma.
GEORGE FRANCIS SUKER, M.D. 100

Iridotasis in Glaucoma.
EDWARD STIEREN, M.D. 108

Report of a Case of Parinaud's Conjunctivitis.
HOWARD V. DUTROW, M.D. 115

The Practical Value and Limitations of the Tonometer.
EDWARD JACKSON, M.D. 121

Conjunctivitis Tularensis (Squirrel Plague Conjunctivitis).
FREDERICK W. LAMB, M.D. 135

Glaucoma Simplex Without Perceptible Rise in Tension.
HARRY S. GRADLE, M.D. 139

Congenital Partial Defect of Retinal Pigment Layer of Iris in Both Eyes.
M. FEINGOLD, M.D. 151

Scleral Trephining in Detachment of the Retina.
JOHN GREEN, JR., M.D. 157

Pressure Changes in the Curvature of the Cornea Due to Chalazion and Other Lid Tumors.
T. W. MOORE, M.D. 169

PAGE

Deviation Upward of the One Eye, Due to Overaction of the Inferior Oblique, Consecutive to Congenital Paresis (or Insufficiency) of the Superior Rectus of the Other Eye.
FRANK C. TODD, M.D. 172

The Use of Eyes of Kittens in Ophthalmic Operative Teaching.
WILLIAM A. FISHER, M.D. 177

Treatment of Cataract at the Central Branch of the National Military Home.
J. W. MILLETTE, M.D. 188

Lantern Slide Demonstration of Roentgenographic Findings of Blind Dental Abscesses Causing Accommodative Asthenopia.
C. B. FULKERSON, M.D. 196

OTO-LARYNGOLOGY

TABLE OF CONTENTS

PAGE

Should the Otolaryngologist Enlarge His Field Diagnostically, Technically, or Both?
T. E. Carmody, M.D. 207

Importance of the Internal Secretions in Ear, Nose and Throat Affections, with Special Reference to the Hypophysis.
Harry L. Pollock, M.D. 211

Relation of the Glands of Internal Secretion to Otolaryngology.
Joseph C. Beck, M.D. 220

Nature and Control of Hemorrhage in Nasopharyngeal Operations.
Joseph Weinstein, M.D. 227

The Interpretation of Stereo-Roentgenograms of the Mastoid.
J. M. Ingersoll, M.D..... 241

The Surgery of the Temporomandibular Articulation.
L. W. Dean, M.D.—W. F. Boiler, M.D..... 247

Problems of the Deaf.
M. A. Goldstein, M.D. 257

Frontal Sinusitis as a Probable Cause of Acute Nephritis.
George F. Keiper, M.D..... 267

Acute Otitis Media of Diphtheretic Type.
William C. Bane, M.D. 270

The Safeguard Against the Radical Mastoid Operation, That Is Early Drainage Through the Mastoid Antrum.
Secord H. Large, M.D. 275

The Value of Bone and Cartilage Transplants in Rhinological Surgery.
William Wesley Carter, M.D. 286

Primary Carcinoma of the Middle Ear, with Report of Case.
Horace Newhart, M.D. 300

Osteomyelitis of the Skull.
H. A. Beaudoux, M.D. 315

The Tonsils as an Atrium of Infection in Poliomyelitis.
Ernest M. Seydell, M.D. 324

The Technic in Tonsil Operation.
Austin A. Hayden, M.D. 331

A Modern Method of Tonsillectomy.
G. E. Gwinn, M.D. 353

EXHIBITION OF INSTRUMENTS

New Use of Michel Clip.
H. L. Pollock, M.D..... 361

A New Laryngeal Snare.
T. S. Blakesley, M.D. 362

PAGE

A New Palate Elevator.
T. S. Blakesley, M.D. 363

A New Ether-Suction Apparatus.
Sidney Israel, M.D. 364

A New Instrument for Attachment to Suction Apparatus to Maintain a Dry Field in Tonsillectomy Under General Anesthesia, and to Lessen Occurrence of Postoperative Pneumonia.
Sidney Israel, M.D. 365

The Tonsillectome.
Frederick W. Lamb, M.D. 367

DISCUSSION BY MEMBERS

Adams, C. F. 352
Andrews, A. H. 322, 351
Avondino, Miss 263, 264
Bane, William C. 274, 359
Bannister, J. M. 186
Beaudoux, Henry A. 322, 323, 351, 352
Beck, Joseph C. 11, 244, 245, 283, 298, 313, 321, 322, 329, 358, 360
Brown, John E. 282
Calhoun, F. Phinizy 93
Carmody, T. E. 204, 255, 256, 274, 322, 352, 360
Carter, W. W. 298, 299, 358, 359, 360
Cloud, James H. 259, 260, 261
Case, George M. 284, 285
Dorsey, J. G. 194
Fagin, Robert 122
Faith, Thomas 10
Feingold, Marcus 37, 147, 155, 156
Fisher, William A. 186, 187, 192, 193
Fulkerson, C. B. 204
Goldstein, M. A. 264, 265, 266
Gradle, Harry S. 8, 22, 91, 92, 99, 132, 133, 150, 155
Green, John, Jr. 73, 107, 167, 168, 176
Gwinn, G. E. 360
Hardy, William F. 38
Hayden, Austin A. 352
Hilliard, Miss Ethel 261, 262, 263
Ingersoll, J. M. 245, 246, 274, 359
Jackson, Edward 58, 72, 133, 134, 149
Jones, E. L. 9, 165
Keiper, George F. 59
Kneedler, G. C. 284, 313, 359, 360
Krebs, Adolph 114
Lamb, F. W. 193
Lamb, Robert Scott 11, 58, 147, 166, 195

PAGE
Loeb, Hanan 10
Lokey, H. M. 93
Lancaster, W. B. 98, 107, 148, 166, 193
Large, Secord H. 285
Lichtenberg, Joseph 73
Mackenzie, George W. 38
Mann, R. H. T. 107
McHenry, D. D. 274
Michie, W. T. 283, 284
Middleton, A. B. 351, 358
Millette, J. W. 195
Murphy, John W. 273, 281, 282
Newhart, Horace 283, 313, 314
Parker, Walter R. 167, 194, 195
Peters, Luther 23, 92
Pollock, Harry L. 322, 359
Patton, James M. 99, 107, 122, 186
Pratt, J. A. 330
Ray, J. M. 171
Reber, Wendell 8, 24, 99, 133, 148, 176, 193
Sawtell, J. E. 350, 351
Seydell, Ernest M. 330
Spencer, F. R. 73, 273
Steuben, F. G. 186, 195
Stieren, Edward 203, 204
Stucky, J. A. 282, 283
Suker, George F. 107, 147, 166
Todd, Frank 176, 312
Vinsonhaeler, Frank 274
Von Colditz, Thomas M. 166, 359
Weil, A. I. 273, 298
Wilder, William H. 164, 165
Williams, Wright C. 147
Woods, Hiram 73, 98, 99, 149, 150, 195
Wright, H. R. 93, 194
Wright, J. W. 194
Young, H. B. 9, 259

PRESIDENT'S ADDRESS

JOHN EDWIN BROWN, M.D.
COLUMBUS, OHIO

It is generally accepted that the address of your president may be as brief as he chooses to make it, and need not necessarily deal with a technical or scientific topic, such as is appropriate for the other papers submitted in our annual program. If there is a keynote to be sounded on the work of the Academy this is the occasion.

My immediate predecessor in office, Dr. Joseph C. Beck, gave us an exposition of the value of what he denominated team work in the practical clinical side of our profession, both for its scientific value and for its commercial adaptability. In his address, Dr. Beck discussed the relationship of otolaryngology to other specialties; while Dr. Lamb dealt with ophthalmology in relation to other specialties, taking perhaps a more technical and scientific view. These valuable addresses and those of others, who have more signally honored than can I the position to which your good will has elected me, make it more difficult to invite your attention to an entirely new phase of ophthalmologic or otolaryngologic interest.

It cannot have escaped your notice that in the two decades which mark the active life of our Academy, we have seen such remarkable changes, not only in the practice of medicine due to the development of its supporting sciences, pathology, bacteriology, serology, preventive medicine, and surgery, but also in the legal and sociologic status of the profession, that they must be regarded as greater than have taken place in any other equal period of time. This has not been because of what our Academy has done; but a practically new organization could not in such a period be simply marking time, as can often be the case with older organizations. Our Academy must have taken to itself the spirit of the time, for during the past twenty years it has grown to be the largest body of physicians engaged in the special work indicated by its name. We, therefore, must be permitted to rejoice in these achievements of a general nature, which include: the increased entrance requirements for medical schools; the necessary increase in all-time teachers to

care for the chairs of underlying sciences; the consequent elimination of many private and unendowed schools; the amalgamation and strengthening of those remaining; the enlarged equipment and greater trained forces in hospitals; the better training for specialties and surgery. It is not to be doubted that, even as from previous disasters of war, medicine will be able to show from the present catastrophe in Europe new truths discovered, or old ones better established.

These are things that are to better equip the individual physician for combating human ills; to aid not merely in prolonging life, but in making that life more comfortable, more enjoyable, and more efficient. All this is in keeping with the spirit of service, which is the true spirit of our profession, and which distinguishes it from business and the trades.

On the other hand, a new difficulty has arisen during this same period which is in part the direct result of the progress through which medicine has become more specialized. To many of our people the service for which we have better trained ourselves has become more inaccessible. A "New Yorker" not long since said that living on Manhattan Island was becoming an impossibility except for the very rich or the very poor. Dr. Cabot has more recently said that the best service in medicine is possible only for the very rich or the very poor. The rich can privately command the multiple services of the best specialists, and meet all the fees without hardships; the poor go to our clinics and hospitals, in which they receive the benefit of the same class of service without entailing any fee. The middle class, the large part of our population, now designated commonly in the cartoons as "the consumer," is unable to pay for this as a private service, as do the rich, and yet is above the social level where he feels that he can accept it as a gratuity. They must groan in their endeavors to meet this phase, among many others, of the high cost of living, or accept a professional service meager and probably inefficient. We must even recognize that the optometry question which by legalizing acts in various states has been forced so acutely on our attention, is still in part simply an economic question.

Now it is inevitable in view of the spirit of the present time that the best equipped medical service must be made available to all alike. Some say that before many years this is to come about through state medicine, for proof of which they cite the following: the increasing state aid to medical

education, increased regulatory medical practice acts, including the workmen's compensation act and other similar laws. Others contend that the problem is to be worked out in such a manner as to involve no revolution from the system of private practice now followed.

One thing is plain, however, and that is that, in the future, the man who limits his practice and who therefore, in that sense is a "specialist," must be able to show that he possesses the qualifications and has received the special training that entitles him to appear before the public in this light. We now, therefore, see that the real opportunities for service in our Academy lie not so much in the scientific programs which help to make students and investigators of our members and which give others the benefit of their work, but rather in the several committee activities in which it may be engaged, and which contribute their portion to the progress of medicine and to the welfare of the public. These services are rendered largely through cooperation with other bodies, and some among us do not realize their importance. Your president would bespeak for these committees a more active cooperative interest among our membership.

At this meeting we can well afford to consider whether there may not be new avenues for standing committee work, through which a service can be rendered to medicine and to our people. It may be wise to consider whether now, with our widely represented membership, we should adopt any new standard of eligibility for membership which would be more in keeping with our present day ideas; whether by quarterly or semiannual brief committee reports, printed and sent out to our members, we cannot arouse a much greater activity and increase the desire to cooperate with social service organizations whose work is among the blind, the partly blind, the deaf, the partly deaf, or others who naturally come under our professional care.

We have had a most profitable propaganda on the matter of preventable blindness, and I know personally that much good has been accomplished in my own state through the activities of the several bodies connected with this work. These activities must be continuous, but other agencies need to be set to work if we are to fulfill our obligations to those who have been blinded or partly blinded. For a period of twenty years I have seen the work of our state school in the education of the

blind. These pupils have needed the opportunity first of training themselves in household and domestic duties, so that in the homes to which they later return they can be a helpful factor instead of a burden. Another need is that of special manual training and trade instruction, so that under a protecting organization there would be a market for their labor and its products. Until very recently this condition has not obtained. Now the old idea of a school training limited to the arts, letters, and music is giving way to the ideas which have made Hampton and Tuskegee famous, that of a training which not only develops the pupils mentally, but also puts them in positions where they are productive citizens who would otherwise be dependent. It has been shown that this can be done for the blind as for others who have handicaps. An organized committee of our Academy that would study the conditions surrounding the education of the blind, and the special opportunities given to the partly blind in the public schools of our states, could do much to promote progress in this important field. This is but one of the many questions deserving our interest. In giving ourselves to the scientific program furnished at our twenty-first annual meeting let us also duly consider questions of this nature.

THE INTERNAL SECRETORY SYSTEM AND THE EYE

ROBERT SCOTT LAMB, M.D., F.A.C.S.
WASHINGTON, D.C.

When sending the title of this paper to our president, the object I had in view was to arrange the eye symptoms which are most often indicative of or caused by disorders of certain glands of the internal secretory system, so as to aid, to a greater or less degree, in the diagnosis of glandular oversecretion or insufficiency.

It is possible that at present few symptoms could be so thoroughly identified with imbalance of any particular gland as to be considered pathognomonic of either increased or decreased secretion. As a matter of fact, the imbalance of any one gland is apt to bring about a reaction in another, or even several glands, so that the earliest symptom becomes either combined or confused with other symptoms, the result of pluriglandular secretion, admixing in the blood or the tissues and causing either intensified or antagonistic stimulation. Therefore, there need be no apology for any present inability to clearly define the exact effect of the introduction into the blood of any secretion from any one gland.

Nevertheless, we must endeavor to associate with each known secretion any symptom or symptoms which may be either caused or modified by it. For, as I have elsewhere pointed out, many of the patients suffering from disorders of one or more glands of the internal secretory system, usually consult the oculist for headache as much as ten years before the general manifestations become sufficiently noticeable to call for the advice of their physician; and an early correct diagnosis may save much mental and physical suffering and some invalidism.

It is to this task, then, I set myself, and the statements herein contained summarize the findings resulting from this study, which is of never ending and intensely absorbing interest.

I am sorry to be compelled to epitomize, because an elaborate discussion would serve better in this attempt to make clear

what has been done in the embryology, anatomy, and experimental physiology of the endocrine system; however, I have to take for granted a preliminary survey or previous study of this subject by every one of us.

It is impossible for us to consider the effects of internal secretory imbalance without frequently realizing the close association with the sympathetic system proper and the vagus or autonomic system.

Some secretions stimulate one system, some the other; for instance, the thyroid stimulates the sympathetic and the thymus the autonomic.

When the sympathetic is stimulated slightly and not reinforced or antagonized abnormally, there is a transient dilatation of the pupils, together with a slight retraction of the lids. But when the stimulation is prolonged or reinforced such signs appear as Kocher's or Rosenbach's (Hoeman's) or Stelwag's; and when this reinforcement and stimulation can be sustained for a length of time, or can be multiplied in intensity over a shorter period of time, such symptoms as Gifford's, von Graefe's, or even a typical picture of Graves' disease may occur. Such a condition usually occurs when the adrenals are strong and the sensitization of the plates of Langley is produced by thyroid hypersecretion. In some such cases the pupils may be smaller than would be expected, but this is because of thymus or posterior pituitary secretions introduced into the blood and having an antagonistic action.

In fact, an upset to the imbalance of secretions sufficient to cause such an outward physical appearance as Graves' disease, is bound to be pluriglandular oversecretion, with the secretions from certain glands dominating.

When the vagotonic or autonomic system is stimulated there is an immediate tendency to contracted pupils; narrower than normal palpebral fissures; esophoria; spasm of accommodation, congestion (deep) of ciliary body and chorioretinal and scleral vascular disturbances. The thymus and pituitary favor such conditions. The more chronic inflammations may usually arise in conjunction with vagotonia, but such a condition as simple glaucoma is probably the end-result of gonadal and adrenal insufficiency; whereas acute inflammatory glaucoma is probably the result of a sudden imperative demand on the adrenals for secretion to sustain the body in its attempt to defend itself against shock, such as fear, in the presence of

gonadal and adrenal insufficiency. For, although the secretion is forthcoming for a short period, the inability to continue to supply it causes a precipitate lowering of sympathetic tone and throws the balance under the control of the vagus. There are commonly other predisposing factors present, such as high hyperopic eyeballs and other anatomic abnormalities.

This conception of the etiology is substantiated by the fact that pilocarpin and adrenalin by hypodermic injection overcome the attack and, furthermore, stimulation to the sympathetic is well known to be always beneficial.

In internal secretory imbalance there may be esophoria or exophoria, depending on whether the dominating secretions are stimulating the vagus or the sympathetic.

Changes in vascularization become arterial activity or venous passivity, intraocular as chorioretinitis, acute or chronic, or extraocular as ciliary or conjunctival congestion, bright red or dull garnet red, depending on sympathetic or vagus stimulation.

Disturbances of vision occur. Visual acuity diminshes in either case and the difficulty of finding proper correcting lenses is markedly increased. Such patients often go the rounds for more reasons than one, as their headaches continue. Binocular fixation is hard to maintain and the patient tires easily and asthenopia is progressive. Hemianopsia may occur as in the case of hyperplasia of the anterior pituitary, a bitemporal hemianopsia, yet other disturbances of retinal circulation causing transient subjective symptoms of scotoma, as scintillating scotoma, are doubtless due to abnormal endocrine secretions.

Is it presumption to have suggested so many possible changes in our conception of the etiology of ocular symptoms?

Having studied the literature of experimental physiology relative to substances derived from the endocrine glands, so far as I have been able to obtain it, and having observed closely the therapy of these substances on normal persons and others known to be suffering from disturbed function of one or more glands of this system, I firmly believe it is not presumption. Furthermore, I feel sure, when you consider what I have suggested and contribute whatever information you may derive from your own observations of the influence of these secretions, normal and abnormal, on the eye and its appendages, you may transcend to even higher flights in this expanse of present day romance in medicine.

DISCUSSION

Dr. H. S. Gradle, Chicago: The subject of Dr. Lamb's paper is too broad for general discussion, and I will therefore limit my remarks to one phase of the subject matter embraced, the suggested etiology of glaucoma. The idea of adrenal insufficiency being the cause of glaucoma simplex is one not touched on by previous writers and is something that will bear discussion. I am not prepared to say "yes" or "no". All previous work along this line has been with the thought of adrenalin hypersecretion, with various findings by different men. One of the first was Kleczkowski, who claimed to have found an excess of adrenalin in the blood of thirteen patients afflicted with acute inflammatory glaucoma, all associated with high blood pressure. Others have been unable to corroborate these findings. When we take a normal eye and inject adrenalin locally, or subconjunctivally, it produces practically no effect in man, though in the lower animals and in frogs there is some mydriasis. There must be some predisposing factor to cause the adrenalin to act on the eye. But if the cervical sympathetic in man be damaged or destroyed, then adrenalin, used in any form, will produce a moderate degree of mydriasis. But for this to be applied to acute inflammatory glaucoma, it is necessary to adopt the theory that this disease is due to a mechanical process, a dilated pupil, in an eye predisposed. Rollet and Curtil have shown that adrenalin when used in an inflamed eye produces at times a surprising fall of tension. Rubert further has proved this point and has also shown that the reverse may occur. These results are explained by the influence of the drug on the vessels. Were adrenalin the exciting factor, we should expect an invariable accompaniment of increased blood pressure, and we know that is not always present. There may have been an associated high blood pressure, but the two are not interdependent. In 186 cases that Kraemer studied, he found that the blood pressure corresponded with that of normal individuals of the same age. In view of these facts, I do not think we are justified in accepting the hypothesis that adrenalin is the cause of acute inflammatory glaucoma. But I do not wish to deny a close interrelationship between abnormalities of internal secretions and subsequent disturbances of metabolism and disorders of the eye.

Dr. Wendell Reber, Philadelphia: With reference to the use of thyroid and pituitary, will say I have had several children of diminutive size come under this description. It was suggested it might be well to try a joint administration of the glands referred to, and the results were little short of marvelous. Some young females have seemed to thrive on the use of thyroid in small doses—⅙ gr. The mistake is to give large doses. Four or five have done exceptionally well on this therapy. I cannot say why until we have some test that will tell us something of the specific ferments. As to the low grades of myxedema, girls over 15 come to us in large numbers, having a dry skin, poorly developed eyelids, and other symptoms, and it is perfectly amazing the results they will show on small doses of thyroid.

I desire to speak of a case of hypophysial disease, published by Dr. Peter which, when it first appeared to us, presented the picture as shown here. (Illustrates at board).

Pituitary and thyroid gland were administered. The visual fields were regained after six months. After another period of administration for approximately a year, practically full color and form fields were regained. The man then neglected his feeding and after a year and a half showed a cutting down for form and field, but after a year's feeding regained it again. After an attack of grip he again became neglectful,

and showed an almost obliterated field on the left side and a decided cutting down of the field on the other side. A decompression was advised, but the patient would not consent to it so long as he had any vision left. This simply showed what can be done by this therapy. It should be tried out before resorting to the operative phase.

Dr. E. L. Jones, Cumberland, Md.: I wish to go on record as believing that retinitis pigmentosa, and certain other atrophies of the retina and optic nerve, are induced by deficiency or disorder of the ductless glands.

During the past six years, whenever opportunity has offered, I have put these cases on treatment with thyroid extract. Some cases have been followed up, and some have not, but I believe the results will bear out the assertion that a close dependency exists.

This is a matter that, by the observations of many men from many points of view, is worth trying out, because we know that everything else has been a signal failure. It has been an unanswered question why retinitis pigmentosa should have the symptom of night blindness, and why we sometimes have night blindness without pigmentation. It is my belief that the hyperpigmentation may be due to adrenal deficiency, as exemplified in Addison's disease. The night blindness, as a symptom, is likely due to deficiency of thyroid secretion, and is not acompanied by pigmentation, unless also the adrenals are deficient in function.

Both French and German observers have reported an enormous increase in night blindness among the soldiers in the trenches, there being now many hundreds in place of the few that existed before the war.

We know that the first effect of stimulation is to increase function and that excessive stimulation of an organ leads finally to exhaustion. Crile has beautifully developed and proved the theory of the kinetic drive, wherein the chief primitive instincts of fear, self-preservation, or the battle for life, cause an immense increase in the internal secretions; but, too long continued, they become totally exhausted. Now, I do not believe that many of these cases of trench night blindness will develop retinitis pigmentosa, or that permanent loss of vision will result, for the war will doubtless end before these causes operate long enough to cause more than functional exhaustion, no organic change being likely.

I have used the thyroid extract also in several cases of atrophy of the retina and optic nerve which were not due to retinitis pigmentosa, and when night blindness was not present, and in some cases results have been obtained that I should not have believed possible if I had not seen them. Any one looking into the eyes of some of these patients would say "the patient is blind," and yet very good vision would be present.

In the earliest case of which I have a record, a man aged 42 years, with much pigmentation of the retina had vision much reduced for distance, somewhat contracted visual fields, and ability to read only coarse newspaper print. He was given thyroids to last for one month. He could not be located until six years had elapsed, and his vision, far and near, had increased over 50 per cent., and the visual fields had not contracted farther. In later cases, arsenic and strychnin have been combined with the thyroids. In some earlier cases small doses of iodin were given, which would activate the patient's own thyroid gland.

Dr. H. B. Young, Burlington, Iowa: You will remember that Hertoghe, when in this country several years ago, made the statement that, while myxedema and cretinism were the definite signs of hypothyroidism, they were doubtless its least common manifestations. I recall these three illustrated cases in the past two years:

(1) A young unmarried woman, with persistent headache and asthenopia, normal vision and emmetropia (under cycloplegia). She had gained weight rapidly and lost hair, almost to baldness, in the preceding six months. She improved rapidly under thyroid extract in small doses.

(2) A fairly well-nourished woman of 45, unmarried, had periodic but irregular attacks of left-sided deafness, which of late had been followed by intense vertigo, nausea, and vomiting. Examination revealed principally a point of contact between the left middle turbinate and septum whenever the mucosa was disturbed. To obviate a possible reflex from this contact, I removed the projecting turbinate; and there was little deafness and no vertigo for the next four months. Then, suddenly, both returned in aggravated form. The possibility of somatic fault and specific infection had due consideration; and the results were alike, nil. Small doses of thyroid extract, however, gave prompt relief; and for the past year her work (school teaching) is uninterrupted.

(3) A man approaching 50, retired farmer, not robust, has had right-sided deafness from childhood (traumatic), and for some weeks lately recurrent vertigo, nausea, and vomiting. His physician says, "not alimentary." The ear is dry, extensively cicatricial, without perforation, and negative to caloric test. He got marked relief from the use of thyroid in 1 gr. doses. I believe that in considering these rather mysterious cases we shall find that ductless gland failure plays an important part.

Dr. H. Loeb, St. Louis: I rise to present a case of what appeared to be congenital deaf-mutism. At least, so far as we could find out the child had never been able to hear. The internists who had him in charge determined there was an adrenal deficiency, and on that theory gave the child adrenalin, and after six or eight months he could hear some, and after a year the hearing improved tremendously. We had removed the adenoids without result. Now, after a year or so, we notice a continued improvement in hearing. The adrenalin has been discontinued. The child is bright and hears pretty well, and I just rise to say that in cases of congenital deaf-mutism there may be this adrenalin insufficiency.

Dr. Thomas Faith, Chicago. I would like to go on record with two cases of otosclerosis, both in young people, with all the attending symptoms of middle ear disease, but without any change in the drum. One of these patients that I had been attending for about a year was operated on for chronic appendicitis. The history of this trouble probably extended way back to the time when I had seen the patient on account of the otosclerosis. I did not suggest the work to be done on the appendix, but it was done in the course of treating her general health. The removal of this focus of infection stopped the progress of the otosclerosis and there has been no return.

The other case was a young lady with all the indications of otosclerosis. In the course of caring for her the tonsils were looked over and their removal was not advocated. In six months or a year after I had seen her and given an unfavorable prognosis, another physician advised the removal of the tonsils, and this was carried out on account of tonsillar disease. As a matter of courtesy, she came to see me and reported that her ears had recovered. Pursuing the regular course of examining her, I assured her it was only a trifle better and had made little progress in two and a half years. I have mentioned these cases before and have been criticized and assured this was only a coincidence, but I feel that here we have a possible explanation of the relief of this condition by the removal of the focus of infection.

Dr. Lamb (closing discussion): I want to congratulate Drs. Beck and Pollock on the manner in which they have presented the glands they have gone into, and while they have not taken up the other glands, perhaps those are a little more intangible, and for that reason we may postpone their more careful study until a later date, because they undoubtedly exert considerable influence upon the symptom, lessening it or exaggerating it, as the case may be.

In the point Dr. Gradle brought out he overlooks two things. Adrenal insufficiency usually occurs in persons whose body functions are on the ebb. That is the fundamental feature. In addition to that, the shock which is associated with acute inflammatory glaucoma takes place. Here the stimulation is taken on by the system controlled by the thyroid and suprarenals. If, in the ciliary body, there are still any frank signs of such tendencies, they can be taken care of. This stimulation lasts only a short time. It cannot continue long; consequently there is a slump. On the high tide you have a high blood pressure. With the sudden dilatation of the coronary arteries, the relaxation of the cardiac muscles of the heart goes on. On the ebb tide, a shutting off takes place and everything relaxes. If, after you throw off your symptoms by the tonic effect of pilocarpin, or the stimulating effect of adrenalin, you can control your case. I have had only ten cases, but that shows something. If I have accomplished one thing, I hope it is this, that everyone will see the grave responsibility that rests on us who see so many of these unpleasant cases, whether it is the nose and throat with turgid membrane, the eye with the dilatated pupil, or the flushed face and wide open eye. The Kocher sign indicates that the trouble has passed the first stage of transient, recurrent irritability of the sympathetic system and is becoming fixed. The subject is all too big to encompass in one paper, but I hope what has been said fixes at least your attention on something that has been overlooked for a long time. You have all seen the cases, and if you look over the picture showing the appearance of some of these hyperthyroid cases, you will recognize dozens of these cases that have come to you in the last fifteen years.

Dr. Joseph C. Beck (closing remarks): Dr. Lamb has proved in the closing of the discussion that he knows what he is talking about in regard to the glands of internal secretion, but that is no reflection on Dr. Gradle. I wish Dr. Pollock might answer the point thrown out as to the proper test for the adrenalization.

As to the case that Dr. Reber reports of the patient who recovered without operation, I wish to say that both the doctor and the patient are to be congratulated. It is not usually the case, and when it does happen a recurrence is quite likely. After the operation on the hypophysis the case should have gland feeding. This avoids some of the unpleasant symptoms of hypopituitarism. The feeeding of this glandular extract to the patient will frequently influence the hyperplastic condition of the nasal structures as well as rarefaction of the internal ear, but not in an inflammatory or true suppurative process.

I know that a number of the gentlemen I see present are using adrenalin in otosclerosis. I regret that they did not get up during the discussion and give their results. This is the place to get up and say what you think about it. We are all looking for some way of benefiting these unfortunate deaf patients, whose number is steadily growing, and if we have something that will stay the process we shall be more than satisfied. I would not make these assertions if they could not be backed up by experience.

Dr. Faith hit the nail on the head in the report of his two cases.

Dr. Boot of Chicago reported some sixty cases of otosclerosis in which he performed a tonsillectomy and the patients were benefited. He is inclined to look on otosclerosis as an inflammatory process, in which we do not agree. Our belief is that the removal of the tonsil or any focus of infection influences the otosclerotic process indirectly through the change in the glands of internal secretion.

RING SCOTOMA IN ITS RELATION TO CHOROIDAL AND OPTIC NERVE DISEASE

WENDELL REBER, M.D.
PHILADELPHIA

Ring scotoma as a symptom of retinal disease, and especially pigmentary degeneration of the retina, is a well recognized phenomenon. It is not frequently associated with choroidal disease and is decidedly infrequent as a manifestation of optic nerve disease. Considerable interest, therefore, attaches to the three following case histories:

CASE 1.—T. H., male, aged 25, single, a cigar dealer, was first seen April 20, 1915. Has always enjoyed good health. He had diphtheria at 6 years of age, scarlatina at 8 years, and an infection which, from his description, was probably typhoid, at 12 years of age. He smokes and drinks moderately. He denied ever having had any venereal disease.

About five months ago he noticed that the vision of his left eye was becoming gradually blurred. Four weeks later he consulted an empiric who gave him a plus 0.50 sphere in each eye. After another three weeks, finding his left eye still failing in vision, he consulted his family physician, who ordered a sulphate of zinc solution for his eye. He avers that at this stage of his ocular affection he could still read large print with his left eye, which would equal about 6/30 to 6/60 vision. By the time he first appeared in my service at the Samaritan Hospital, vision in his right eye equalled 5/4, while that of his left eye was only 2/60, and this was eccentrically down and out. The right eye was normal in all respects. The left eye was normal as to the anterior segment. The vitreous was full of stringy opacities and no fundus view was obtainable. With a 10 mm. object, three islands in the field of vision were found in which the object was perceived. The tension in the left eye was 12 mm. (Gradle tonometer).

The patient was admitted to the private ward, and blood was taken at once for serologic tests. The urinalysis was normal, the cardiovascular system also normal. The rhinologist reported that he could find nothing abnormal in the nasal passages or sinuses. The teeth were particularly good. The tonsils were clinically negative. Two days later all the complement fixation tests were reported negative save the Wassermann, which was positive, being $++++$. Diaphoresis and inunctions were begun at once, and one week later the vision equalled 6/60 and the field enlarged. This is of much interest, in that it indicates that central vision had now returned in an area 5 degrees about the fixation point, and two of the islands had merged. Vision now

equalled 6/15. The treatment was continued, and four weeks later the field chart showed a coalescence of all the outlying portions of the field which now surrounded an irregular ring scotoma.

At this time vision in the left eye had risen to 6/9. Two weeks later, this complete ring scotoma had given way above, leaving two small tiny absolute scotomas to mark the site of the previous ring. Vision in the left eye now equalled 6/7 and the ophthalmoscope revealed a cloud of fine vitreous opacities with general roughening of the choroid in the macular region, but no defined lesions. This was on June 3, 1915, about six weeks from the time he was first seen. Wassermann at this time was faintly positive. The patient was allowed to go home with instructions to his family physician to continue mercury and iodids for at least a year. Eight months later he appeared again with a perfectly quiet eye. The vision was 6/6 plus, the choroidal status being about the same as on June 3, 1915. There is still a small narrow absolute incomplete ring scotoma below and two tiny absolute scotomata above.

Remarks.—This was unquestionably a syphilitic choroiditis or chorioretinitis in which the neuroepithelial layer was principally involved. When the vitreous humor had sufficiently cleared to permit accurate ophthalmoscopic inspection, choroidal disturbance sufficient to adequately explain the visual field phenomena was easily detected. Nothing could be more interesting than to watch the visual field reform in its periphery and center under treatment, so that finally all but a tiny rim above the macula was restored to full function. It is also worthy of note that the scotomata which had been positive during the observations of 1915 had become negative when the patient was seen in 1916. This is in accord with the dictum of Dr. Luther Peter in his recent volume on perimetry.

Case 2.—J. A. McA., male, aged 30, proprietor of a barber shop, came complaining of poor vision in his work, especially late in the afternoon. This was first noticed about 10 weeks previous. Was given glasses by an empiric as follows: R. E.: — 25 sph. — 50 cyl. 165 degrees = 6/12; L. E.: — 50 sph. — 50 cyl. 30 degrees = 6/17. No history of any venereal disease obtained. His general health has been good; measles at 6, scarlatina at 7, pertussis at 8 years of age, but no illness since then. He is a total abstainer from alcohol. He is a small but well built, well nourished man who leads a quiet, well regulated life. No clinical history of anything that resembled the symptoms of the secondary stage of syphilis could be elicited. The glasses have not been of any service, and the sight in the right eye has been slowly growing worse. When first seen, his unaided vision was R. E. 6/15; L. E. 6/9, and corrected R. E. 6/12 plus; L. E. 6/7. The accom-

modative power equalled the 0.50 D. type from 6 to 10 inches in the right eye and from 6½ to 18 inches in the left eye.

The ocular movements in all meridians were smoothly and completely executed. The anterior segment was normal in each eye. The pupillary reactions were full and equal and the knee jerks normal. There were 2 degrees of esophoria and no hyperphoria. The ophthalmoscope showed clear media. The optic nerve of the left was decidedly hyperemic, with slight blurring of the entire circumference, particularly on the nasal edge. The outlying fundus was perhaps a trifle hyperemic. The same condition was presented in the left eye ground, but it was not so marked as regards the optic nerve. The visual fields, taken on the following

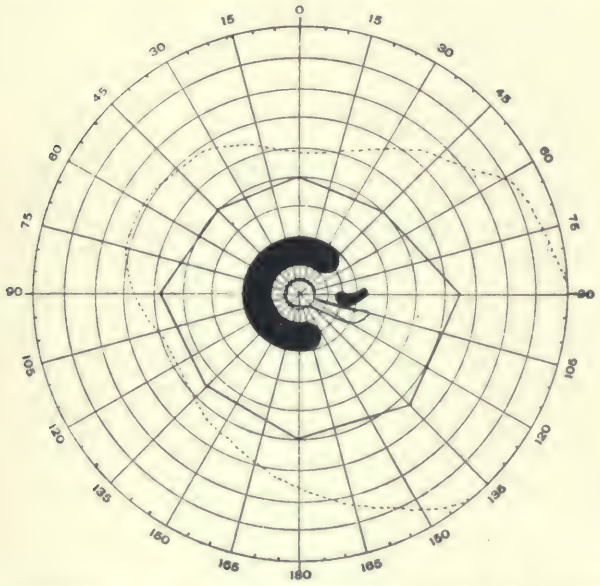


Fig. 1.—December 9, 1913. Form field contracted. Blind spot enlarged. Absolute ring scotoma.

day (Dec. 9, 1913), revealed in the right eye an almost complete ring scotoma. (Fig. 1). There was also concentric contraction of the form fields with noteworthy enlargement of the blind spot of Mariotte. Of much interest was the red field, this being of gourd shape. The head of the gourd occupied the fixation point and the handle reached almost halfway round the lower expanse of the enlarged normal blind spot. This same day the eyeground was studied with especial care under a transient mydriatic because of the interesting visual field phenomena, but no choroido-retinal lesions, or even a suggestion of the same, could be found in the perimacular region, or, for that matter, anywhere in the eyeground other than the optic nerve. Because of the complete absence of any discoverable ophthalmoscopic lesions in the retina and choroid, there was some doubt in my mind about the real

existence of the ring scotoma, and I therefore repeated the perimetric examination, which had been made by my assistant, Dr. Lawrence. The results corresponded at every point with his findings as shown in Figure 1. The left visual field showed some contraction of the form and red fields, but on this side there was absolutely no scotoma or enlargement of Mariotte's spot.

The patient was admitted to the Samaritan Hospital the following day for further study and for treatment. Two days later the internist reported the man as physically normal. Urinalysis was negative. The Wassermann was $++++$, however, and we therefore counseled the use of salvarsan. The drug was used

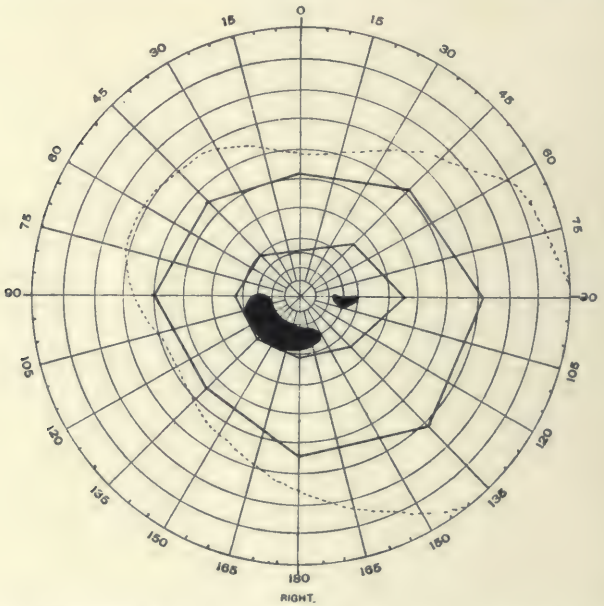


Fig. 2.—December 20, 1913. Blind spot and ring scotoma shrinking under antisyphilitic medication. Ring scotoma absolute.

intravenously and, in addition to this, daily sweats were given with the electric body hood. Improvement began immediately. On December 20 his corrected vision had risen to 6/9 and the ring scotoma was only about two-fifths of its previous size. The optic nerve was of much better hue (Fig. 2). It is also noted that by this date the limits of the red field had assumed more nearly the normal proportions and now were found entirely outside the scotoma.

By December 31, 1913, eleven days later, the corrected vision in the right eye had risen to 6/7 partly; the ring scotoma had diminished to a small spot almost directly below the fixation spot, but there was still an absolute scotoma for white. Mariotte's blind spot was now almost normal in size, as were also the form and red fields (Fig. 3). By this time the hue of the optic nerve

had returned to normal. January 6, 1914, seven days later, the corrected vision in the right eye was 6/6 plus. The visual field chart of this date shows that the previous absolute scotoma for white had become relative, but still remained absolute for red. Mariotte's blind spot was now entirely normal in extent and the form and red fields had filled out to the normal limits (Fig. 4). The refraction at this time equalled: R. E. — 0.37. — 0.37 cyl. axis 170 degrees = 6/6 plus; L. E. — .50 cyl. axis 180 = 6/6. The patient was now returned to the care of his general medical adviser who was enjoined to keep him under spirillicide medication for a year at least.

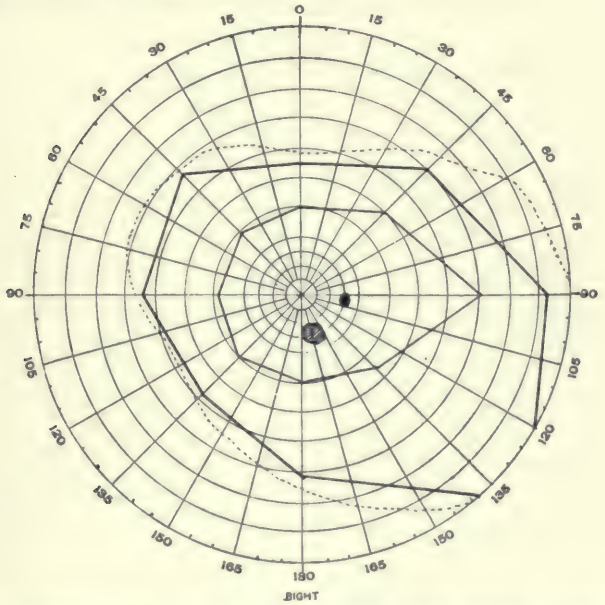


Fig. 3.—January 6, 1914. Form and color fields almost normal. Blind spot of Mariotte about normal. Small relative scotoma—the remains of the ring scotoma.

Remarks.—At the first glance, this case would seem almost a duplicate of Case No. 1. The visual field phenomena were, in many respects, identical. But it must be urged that the ophthalmoscopic changes were reversed. In Case No. 1 there were fine choroidal changes which readily accounted for the visual field defects, the optic nerve showing very little if any alteration. In Case No. 2, however, the clear media permitted most accurate ophthalmoscopic examination of every stage of the malady. At no time could the slightest change be detected in the foveal or perifoveal retina and choroid, while the optic nerve exhibited distinct changes. It must be frankly conceded that there may have existed fine ultra-ophthalmoscopic changes

in the neuro-epithelial layer in this case. Somewhat similar instances are found in those toxic amblyopias that exhibit fine central relative or even absolute scotomas without recognizable ophthalmoscopic changes. True, in such cases, there is impairment of function of the ganglionic elements in the retina; and, if the disease progresses far enough, the lesions emerge from the ultra-ophthalmoscopic class (in the sense that they are not recognizable) to the ophthalmoscopic class that are recognizable. Herein lies the inestimable value of the perimeter, or preferably the campimeter. But in the cases under consideration,

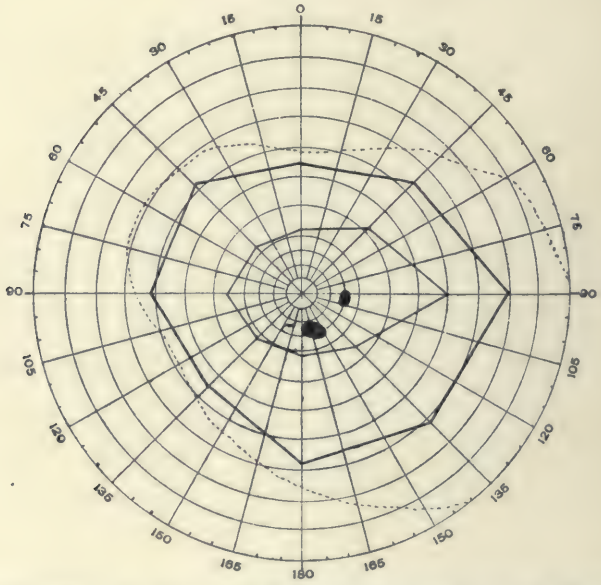


Fig. 4.—December 31, 1913. Form field almost normal. Normal blind spot slightly enlarged. Minute area of ring scotoma left, still absolute.

only the optic nerve was distinctly affected and the question definitely arises: Could there be an optic nerve lesion that might produce such an almost mathematically outlined ring scotoma?

This brings up the whole question of the arrangement of the nerve fibers in the optic nerve and their distribution in the retina, about which there is not as definite information in the literature as one might wish.

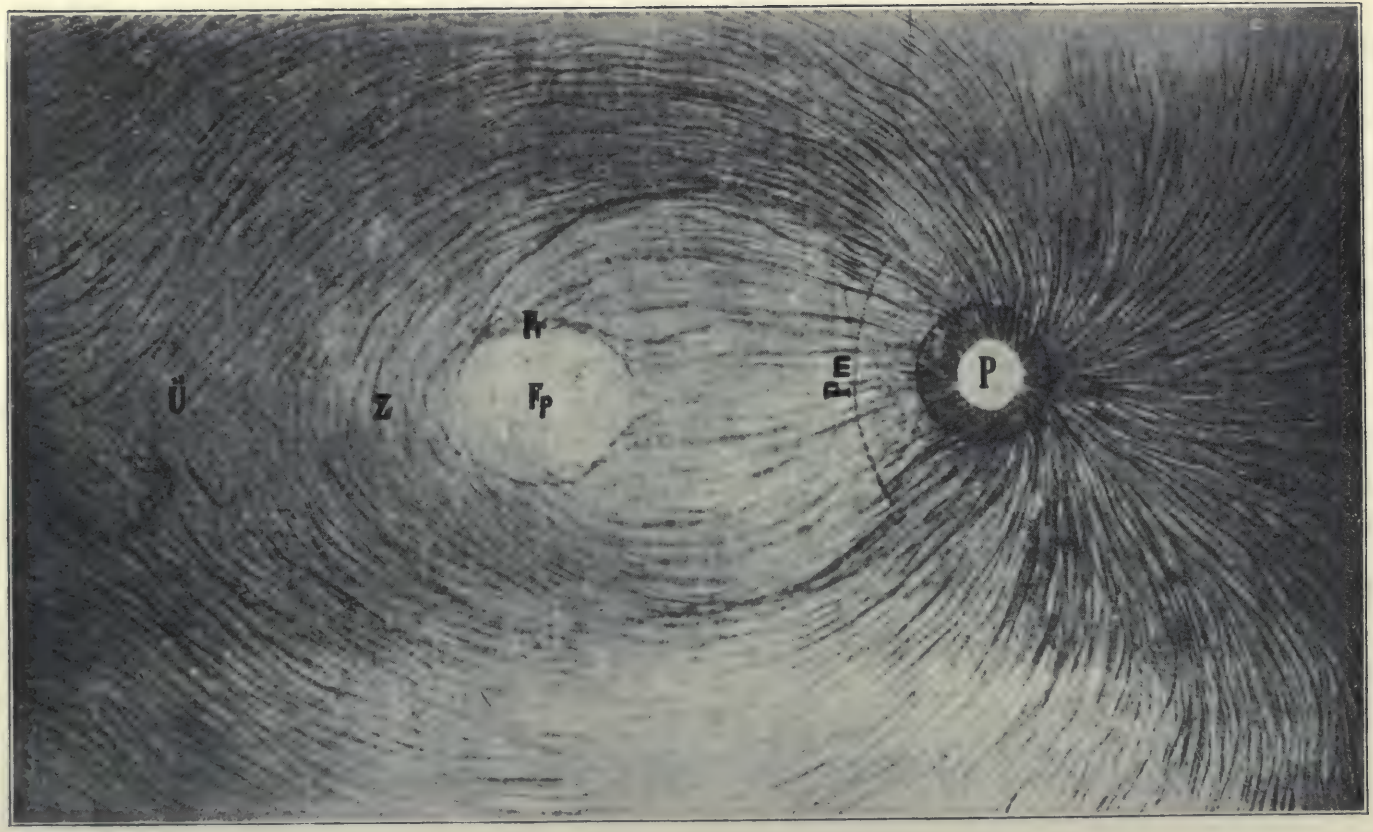
It may be said that the greater number of authorities agree in the general statement laid down by Parsons that the fibers from the peripheral or anteriormost parts of the retina are to be found in the middle of the optic nerve, while the fibers from

the area of the retina nearest to and surrounding the papilla are to be found in the peripheral portions of the optic nerve. (A glance at Figure 5, which is grossly diagrammatic, will illustrate what is meant by this statement. Some prefer the statement of Fuchs that "the fibers of the optic nerve upon entering the interior of the eye spread out like a sheaf to form the most internal (most anterior) layer of the retina. The fibers which constitute the margin of the papilla terminate in the vicinity of the latter. The nearer the fibers lie to the axis of the optic nerve, the greater is the distance that they have to traverse in the retina in order to reach the level of the next layer of the retina, the ganglion cell layer, with which they are continuous. Accordingly, the following may be enunciated as representing the state of the case: Those fibers which come from the peripheral portions of the retina lie in the center of the optic nerve, while those which rise from the central regions of the retina lie along the margin of the nerve." To return once more to Parsons: "These fibers probably maintain this relative position as far back as the chiasm. The only exception applies to the papullo-macular bundle. These fibers pass into the outer part of the nerve where they are spread over an area which is triangular in section with the apex toward the center of the nerve."

Illustrations of such longitudinal sections of the optic nerve and retina as are to be found in the literature give only gross and vague ideas as to the exact method by which the optic nerve fibers are distributed to the various parts of the retina. If we attempt then to explain the presence of a ring scotoma by a lesion in the fibers of the optic nerve, we find no exact basis on which to explain it. A close study of the relative position of the fibers as revealed by a cross section of the optic nerve throws no light on the possible location of such a supposed lesion. On the other hand, if we examine the scheme of Michel[1] as to the arrangement of the optic nerve fibers in the retina, there is bare possibility that a localized edema, or even grosser lesion of the optic nerve fibers just around the papillo-macular bundle might compromise the percipient elements in the retina supplied by these fibers.

This is a pretty fine assumption we must admit, and the possibility of such a perfectly defined lesion in the optic nerve is remote indeed; the more so when we reflect upon the well-

1. Graefe Saemisch, Handb. d. ges. Augenh., 2d Edition.


Ü
Z
F_r
F_p
E
P

known greater vulnerability of the papillomacular bundle. Taking all the possibilities of the case into consideration it is decidedly more probable that there may have been a localized toxic edema of the neuro-epithelial layer surrounding the macula.

As is well known, the retina is loosely attached to the choroid, except at the optic nerve entrance, the ora serrata, and the macula. The conception, therefore, of an edema of the neuro-epithelial layer surrounding the macula but not invading it (because the retina and choroid are closely attached to the macula) does no violence to our present ideas concerning the nervous and vascular anatomy of these parts. As de Schweinitz aptly says in his article on "Homonymous Crescentic Scotomas in Association with Ethmoiditis and Tooth-Root Abscess,"[2] the attempet to definitely localize such an area in the optic nerve "becomes pure guess work." Side by side with this statement, however, must be placed the statement of Wilbrand and Sanger,[3] who admit that the explanation of ring scotoma on any basis is met with many difficulties.

The one form of optic nerve disease in which complete or incomplete ring scotoma may be encountered is that found with multiple sclerosis. The following case history is therefore not without interest:

Case 3.—H. S., male, aged 39, married, a newspaper man, states that about three years ago he first noticed a strabismus affecting principally his right eye. He paid no attention to it, as he was deeply interested in some business affairs at the time. The accompanying diplopia gave him great annoyance for a while; but, after wearing a patch over his eye for three or four months, he found he could go without it and suffered no further inconvenience. He came principally because of asthenopia in near work. The corrected vision was as follows: R. E. plus 75 cyl. axis 30 degrees = 6/6; L. E. plus 50 cyl. axis 180 degrees = 6/6.

There was insufficiency of the right external rectus, the temporal rotation in the right eye being but 25 degrees. All the other rotations were practically normal; but all were decidedly nystagmic as the limits of rotation were neared (intention tremor). This was beautifully illustrated when the rotations were studied with the tropometer. With the ophthalmometer each eye individually showed a fine lateral nystagmus, when the patient was asked to look steadily into the tube of the instrument. The patient's gait was typically that of multiple sclerosis, and he presented also scanning speech. There was a fine hand tremor on

2. Ophthalmic Record, April, 1915.
3. Die Neurologie des Auges, iv, 40.

both sides. His accommodation was normal and equal in both eyes. There was no longer any diplopia in any part of the binocular motor field, as he had arrived at the period when the brain had learned to suppress the image from the right eye. Both optic nerves were quite pallid throughout. Indeed, their appearance was in striking contrast to the normal vision secured with his refractive correction. The outlying fundus was absolutely normal, save for a rather marked beginning angiosclerosis of the retinal vessels.

The visual fields exhibit an almost complete but irregular ring scotoma in the right eye. Indeed the fields are strikingly similar to the case reported by Wilbrand and Sänger.[4] I saw the patient but twice, as he was about to remove to a western city. The neurologic diagnosis was confirmed by a neurologist. Uhthoff[5] chronicles a similar case in which there was variation in the fields from time to time, as there was also in Wilbrand and Sänger's case. As Spiller[6] points out, the optic atrophy in multiple sclerosis is seldom complete, but tends towards islands of degeneration here and there. This will account also for the apparent discrepancy between the ophthalmoscopic picture and the vision; also the visual fields.

This case is one of frank optic nerve disease accompanied with ring scotoma. If the lesion is such, and a case could be studied postmortem in its relation to the visual field phenomena antemortem, we should come into possession of some precious data. They might perhaps throw some light on the possible location of the lesion in a case like No. 2 above detailed.

Enough has been said to indicate graphically the scantiness of our knowledge concerning the exact and accurate nature of the changes underlying ring scotoma. That it is choroidal in origin in the vast majority of cases is perfectly plain; but we can at present only speculate on just where the lesion is and just what its nature. There can be no dissent from the statement that this is a phenomenon worthy of our earnest study.

DISCUSSION

Dr. H. S. Gradle, Chicago: I have not had much experience with ring scotoma in connection with accessory sinus disease; but during the past year and a half I have had the pleasure of seeing two, one of them absolute and the other relative. No ophthalmoscopic lesions visible. When I last saw them a month ago the scotomata were still present, but were only relative. There is a question in my mind as to their resulting from sinus disease. I would believe that an extension of a sinus disease

4. iv, 430.
5. Arch. f. Psychiat., xxi, 1, 2.
6. Posey and Spiller, p. 470.

would manifest itself first in some other form than ring scotoma, possibly by enlargement of the blind spot. Dr. Reber's explanation of the fibrous form of the nerve will show what occurs. It is undoubtedly by way of the soft tissues. It extends from the sinus through the orbital wall and affects the periosteum of the orbit. At the entrance of the optic canal, the dura and the periosteum blend into one membrane, from which small vesicles penetrate the nerve to enter the central vein of Kuhnt and Vossius. In all likelihood any trouble focuses upon this point.

Here any pressure would have to be presumed primarily on the nerve, and from an anatomical standpoint it would be the fibers at the periphery of the optic nerve that are affected. Hence any pressure would result promptly in an enlargement of the blind spot. Physiologically speaking, these fibers are somewhat resistant and there may be cases in which the pressure may be transmitted through the entire nerve. The least resistant fibers are the papillo-macula and that would result not in a ring scotoma but in an absolute central scotoma. This would be the first manifestation of accessory sinus disease, but I cannot agree that every case can be diagnosed by this means. Furthermore, it has been my experience that it is not the acute suppurative forms that cause this trouble, but rather the hyperplastic cases that manifest themselves in the enlargement of the blind spot and in some cases as a central scotoma.

Dr. Luther Peters, Philadelphia: The subjective ring scotoma is an interesting one. Relatively it is rather infrequently found, largely because perimetric studies are instituted rather late. Ring scotoma, I believe, is an early phenomenon, and for this reason often overlooked. The etiology is, as Dr. Reber has stated, *sub judice* and admits of much speculation. Until the arrangement and course of the axis cylinders of the nerve fiber layer of the retina are definitely determined and the position of these fibers in the optic papilla and nerve is definitely known, we are forced largely to the conclusion that a ring scotoma is a product of disease of the choriocapillaris. We must admit the possibility of at least a partial ring scotoma in certain diseased conditions of the optic nerve proper, as for example, in multiple sclerosis; and in view of our imperfect knowledge of the distribution of the nerve fiber layer of the retina, the same, from clinical evidence, must be admitted in disease of the retina. It is, however, contrary to the usual sequence of development in disease of the optic nerve and retina.

Clinically and pathologically, disease of the choroid advances from foci scattered over the choriocapillaris, whereas disease of the retina is more or less diffuse and advances in a more or less concentric fashion. This advancement is due to the difference of the plan of nourishment of the neuro-epithelial layer of the retina by the choroid and the ganglionic and nerve fiber layers of the retina, the latter being nourished by the central artery and its branches. As most forms of disease advance along the course of the blood streams, it becomes an easy matter to explain the formation of the ring scotoma in choroidal disease, and exceedingly difficult to explain it in diseases of the retina. Uhthoff found at least two cases of multiple sclerosis in which changes were observed in the choroid. The relation, too, of the retina and the choroid is so intimate that disease of the one is usually associated with disease of the other. In retinal disease, therefore, a ring scotoma in many instances is due to involvement of the choroid. This is particularly true in retinitis pigmentosa. More careful studies of the color behavior, and particularly that of blue, in these scotomatous areas may help to solve the problem. A scotoma for blue with less involvement of the red and green suggests choroidal disease.

In the light of our present knowledge, I am inclined to regard a complete ring scotoma as evidence of choroidal disturbance primarily. If the scotoma is formed from distinct foci which finally coalesce, or recovery takes place by the breaking up of the ring into distinct islands, the lesion is probably in the choroid. If absolute in character, also the blind area is probably of choroidal origin. A positive scotoma rarely occurs in disease of other parts of the visual path. When associated with peripheral field changes, an incomplete or partial ring scotoma, indistinct or relative for one or more colors, may be caused by disease of the ganglionic layer of the retina; possibly, but very doubtfully, by disease of the nerve fiber layer, or of the optic nerve.

Dr. Reber (closing discussion): One point; it would be easy to overlook the ring scotoma of this man's right eye, as his central vision was fairly well preserved. The second is the stumbling block of the enlarged blind spot. Without that I should be disposed to put it down as a choroidal lesion. I agree with Dr. Gradle that it is a late form and not the early acute form that causes the hyperplastic condition.

"RENAL CHOKED DISK"

WILLIAM F. HARDY, M.D.
ST. LOUIS, MO.

The title of this paper was perhaps ill chosen, but was selected for reasons of brevity and euphony. It is extremely difficult to limit oneself in discussing such a subject having so many ramifications. Throughout the paper the terms "optic neurititis" and "papillitis" will be employed interchangeably, while "choked disk" and "papilledema" will be used as synonyms. A perusal of various textbooks shows that virtually all employ the terms optic neuritis, choked disk, papillitis, and papilledema interchangeably. A better nomenclature is needed. The same thought has been expressed by de Schweinitz and Holloway[1] in their paper on the operative treatment of papilledema. They state that the terms "optic neuritis," "descending neuritis," and "papillitis" give rise to erroneous impressions and should be abandoned. "Tumor papillitis" and "brain tumor optic neuritis" are objected to as incorrect; choked disk and papilledema are considered synonyms and are to be preferred. In like manner objection should be made to such terms as "albuminuric neuritis and retinitis;" they should be dropped. The prefix "albuminuric" is manifestly improper. Nephritic, renal, or toxic neuritis and retinitis have been suggested as being better. Albuminuria is but a symptom in nephritis. It may or may not be present. No one hears of glycosuric coma or glycosuric retinitis.

At the outset, I shall probably invite criticism by aligning myself with those who definitely discriminate and distinguish between optic neuritis and choked disk. In common with others, it is my belief that papilledema is primarily a non-inflammatory condition. If in its later stages there be found histologically an infiltration with round cells, this finding is a consecutive and secondary change. It is the result and not the cause of the choked disk.

The contentions embodied in this paper, and made a prelude to the direct discussion of renal choked disk, are: (1) That optic neuritis and choked disk are etiologically distinct entities;

1. Trans. A.M.A., 1909.

(2) that the assumption of a manifold pathogenesis of choked disk is probably incorrect; (3) that papilledema is primarily of mechanical origin, all the concomitant phenomena being accountable for on that basis; (4) that the inability to always clinically distinguish between the two conditions does not necessarily imply a common origin or identical process; (5) that optic neuritis is primarily and essentially an inflammatory process. Choked disk is primarily and essentially an edema and lymph stasis, the resultant of increased intracranial tension; (6) renal choked disk exemplifies both processes, each with its separate pathogenesis, showing a lymph stasis with edema and also inflammatory changes.

In discussing renal choked disk it is imperative that something be said of choked disk in general. The multitude and diversity of opinions regarding this condition precludes an exhaustive discussion in the present paper. Roughly they may be separated into two classes: (1) those attributing the process to increased intracranial tension; and (2) to inflammation. All other theories are but modifications and variations of these. Histologic studies must, to a great extent, form the basis of theories and opinions. In studying choked disk, it is necessary to get eyes immediately after death, as edema is apt to be modified or influenced, or, in the case of the nerve, even to be simulated, by the process of fixation.[2] Artefacts are also likely to be regarded as pathologic manifestations in the optic nerve.[3] Paton and Holmes[4] in a microscopic study of fifty eyes from cases of tumor papilledema concluded that the histologic characters in any stage of the process are not inflammatory. On the other hand, there may exist a true neuritis, even eventuating in atrophy, in which there is ophthalmoscopically no appreciable edema or swelling of the nerve head. In the atrophy following a papilledema it is not necessary to invoke an inflammatory process to account for it. According to Parsons, it is a neuritic degeneration. Cushing[5] has definitely stated that choked disk is an edematous swelling of the optic papilla and that the final round cell infiltration and atrophy are not the result of an inflammatory process as the term "optic neuritis" would indicate.

2. Paton and Holmes.
3. Parsons.
4. Brain, xxxiii.
5. Surg., Gynec. and Obst., 1905, p. 298.

On numerous occasions a choked disk has been known to subside completely in a few hours after a palliative operation. This observation is not compatible with an inflammatory theory of choked disk. Optic neuritis, i. e., a true inflammation, results from something more than blood and lymph stasis. Bacterial or toxic agencies must be at the bottom of most cases of a true neuritis. To my mind, blood and lymph stasis in the optic nerve no more constitute a neuritis than blood and lymph stasis in the legs of a decompensated heart patient constitute cellulitis. It has been well said that "inflammation is the reaction apposite to injury." Consequently, long continued irritation of the fluid of edema is expected to result in tissue changes.

It is common to meet with descriptions and accounts in which the terms optic neuritis and choked disk are used at random, or else to designate a mild or severe affection. The impression is frequently conveyed that a given cause operates to produce at one time an optic neuritis and at another time a choked disk. It is to be inferred from such statements that optic neuritis, papillitis, papilledema, and choked disk are synonymous terms for processes which differ only in degree. In view of the fact that microscopic examinations of true choked disk have shown it to be noninflammatory; also that it can be produced almost at will experimentally by increasing intracranial pressure, and finally that reduction of tension by decompression will often result in a short time in a rapid flattening of the swollen disk, makes the identity of choked disk and optic neuritis improbable.

In Clifford Allbutt's book[6] the author writes at length to point out the difference between optic neuritis and choked disk. The latter he designates "ischemia papillae." To quote his own words, he says: "Ischemia of the disks (choked disk) may often, but cannot always, be distinguished with the mirror from optic neuritis, as the two are frequently associated." In increased intracranial tension all the cranial nerves are subjected to the heightened fluid pressure; but they do not as a rule develop a neuritis, even when there exists a toxicity of the cerebrospinal fluid.

In an article dealing with the involvement of the cranial nerves in cerebellar tumor, Grey[7] states that Muskens' studies

6. The Use of the Ophthalmoscope, 1871, p. 55.
7. Johns Hopkins Med. Bull., Sept., 1916.

have determined that disturbances of the olfactory sense in patients, with increased intracranial tension, are of frequent occurrence. This has been attributed to pressure of the third ventricle; but Mills and others have suggested that anosmia may result from nerve choking similar to that which occurs in choked disk. Fuchs and von Graefe have suggested that choked disk can appear without inflammation; it need not be a true neuritis. Jacobson is credited by these writers with having first shown this. Michel gave a differential classification of optic neuritis and choked disk based on the anatomico-pathologic as well as on the clinical aspects of the conditions. Following him, Adam[8] distinguished between optic neuritis and choked disk. According to Adam, the most essential points of differences are: (1) The behavior of the vessels. In choked disk there is a considerable difference in the fullness of the veins and the arteries. The veins are distended, the arteries contracted, while in optic neuritis the arteries are almost normal and the veins overfilled. (2) The elevation of the nerve head in choked disk is usually more than 3 D. above the retina, while a papillitis seldom reaches such a height. (3) The behavior of vision. In optic neuritis the vision is usually much impaired at a very early stage (central scotoma), whereas in choked disk it may remain nearly or quite normal for a long time. In early tumor choked disk the optic nerves are subjected to a fluid pressure, except in unusual instances in which the tumor presses directly on the nerve and causes atrophy, not papilledema. This fluid pressure is not great enough to block nerve impulses; consequently vision is not greatly impaired. Compression of the nerve substance in any marked degree would produce a central scotoma, a rare finding in choked disk.

While many look upon optic neuritis as the same process as papilledema in minor degree, Sattler[9] takes the reverse position and considers optic neuritis as the accentuated expression of papilledema. The view that a convexity forward of the lamina cribrosa marks the dividing line between papilledema and optic neuritis is without confirmation. The posterior fibers in choked disk are rarely bowed forward; but the anterior fibers are usually widely separated and arched forward.

Time does not permit of a discussion of the white areas in renal retinitis, of the macular star figure, the composition

8. Ophthalmoscopic Diagnosis, 1913.
9. Jour. Ophth. and Oto-Laryngol., Aug., 1915.

of the exudate, or of the similar appearing and often confusing lesions in tumor choked disk. In the last mentioned case it may be impossible to differentiate solely from the ophthalmoscopic picture.

Changes simulating renal neuroretinitis, with or without choked disk may appear in patients with no nephritis whatever. Ocular changes ocur in approximately 25 per cent. of cases of nephritis. There are no exact figures regarding renal choked disk. Few cases have been described, and fewer still in which a histologic examination has been made. The confusion in the use of designating terms may account for the paucity of case reports, the affection being no doubt usually classified as an albuminuric neuroretinitis. Renal choked disk occurs as a rule in chronic nephritis. In one of Kampherstein's cases bilateral papilledema occurred in acute nephritis. The prominence of the papilla is said to be usually not great, although at times it is as much as is commonly observed in brain tumor. In two cases seen by me in the past year the height of the swelling in each instance exceeded 5 D. The age of the patients varies; but the trouble occurs most frequently in the relatively young or middle aged. The blood pressure is usually very high, being over 235 in both my cases.

It is a nice point to decide whether the excessive blood pressure is related in cause or effect to the heightened intracranial tension. From experimental evidence we know that it is only by the rise of blood pressure to a height greater than intracranial tension that life is maintained, and when this fails the animal dies. The prognosis is exceedingly bad. In one of the above cases death occurred in two months and in the other in eight months from the time I first observed them. Both had uremic attacks.

Forster stated that nephritics with choked disk were doomed to die in a brief time, and that their mode of exit was with uremia. Kampherstein contended that renal papilledema is in general an indication of a simultaneously existing brain affection, or at least an indication of increased intracranial pressure. This view was probably based to some extent on the postmortem findings in a case of Schmidt-Rimpler's in which, in a girl aged 10, who died in sixteen days after coming under observation and who had albuminuria and high grade choked disk, a pachymeningitis hemorrhagica was found.

Renal choked disk is not confined to human beings. Schlampp[10] recorded the occurrence of a double sided papilledema with great defect of vision and almost inactive pupils in a 10-year-old dropsical dog, in which postmortem examination revealed the presence of much shrunken kidneys and other evidence of renal disease.

An interesting and rare condition was observed by Raubitschek and reported by him.[11] In a patient with a nephritis there occurred an alternating papilledema. A pure optic neuritis in renal disease is a rather infrequent finding. A genuine papilledema is even rarer; but the observation of Raubitschek must be considered unique. His case showed an alternating character, as the right eye when first seen was normal and the left had papilledema with hemorrhages and vascular congestion. In two weeks the papilledema had disappeared, leaving the disk pale and vessels much constricted. A week later the right disk became swollen and, in a few days more, there was present a well marked choked disk. In commenting on this case the question was raised as to whether a predominant part was to be ascribed to arteriosclerosis per se, or to the nephritis. It is impossible in many cases to differentiate in this regard owing to the close relationship of the two conditions. One can readily understand the production of hemorrhages due to high blood pressure, but not the production of a choked disk from the same cause. Pressure of a rigid artery might compress the vein and thereby produce a venous engorgement; but that would not explain lymph stasis, which is a necessary concomitant in producing choked disk. The cases of renal choked disk are, however, found in those patients showing the higher blood pressures. Old age has seemingly little to do with it, as renal papilledema may appear in the relatively young.

Raubitschek briefly reported a second case, in the same communication, in which there was an alternation of the papilledema, but with an interval of six months or so between. The author thought that his findings controverted to some extent the contention of Kampherstein, who held the opinion, as noted above, that choked disk in nephritis is an indicator of an associated disease of the brain, or at least of heightened intracranial pressure. Regarding this, Coats, commenting on Raubitschek's case, stated that it is not difficult to understand

10. Arch. f. vergleich. Augenh., ii, 120.
11. Klin. Monatsbl. f. Augenh., March-April, 1914.

how nephritis might lead to increased intracranial pressure; but that it is not easy to see why the resulting papilledema should be unilateral. A similar unilateral incidence is, however, sometimes observed in cases of cerebral tumor. This latter fact is well known and has been explained as being due to the mechanical or other shutting off of one vaginal nerve sheath, the choked disk appearing in the other eye.

The rôle of diminished urea output raises a question. Does it cause a rise in intracranial tension? Is there a chemical explanation? This has been answered in the affirmative, but mainly in the negative. Cannon considered it is probable that edema of the brain, particularly after injury, may be due to the biologic law that tissues deprived of oxygen take up fluid. If this be true, the manner in which the brain is deprived of oxygen, whether acute or chronic, should not affect the general conclusion. It is fairly well established that the oxygen carrying power of the blood in nephritis is reduced; therefore this might be ventured as a possible explanation for an edema of the brain in chronic renal inflammation.

One can readily understand the symptoms generally accompanying choked disk (vertigo, headache, vomiting), as the result of a common cause, viz: increased intracranial tension. Advocates of the inflammatory theory must experience difficulty in explaining these phenomena without invoking some other factor. On an increased tension basis the headaches, stupor, vomiting, vertigo, and coma of nephritis and of renal papilledema are readily intelligible. Traube in 1861 promulgated some such theory. He held that the symptoms of uremia are caused by cerebral edema. This theory was abandoned and then revived by Byrom Bramwell, Russell and others. Cushing and Bordley[12] inclined to that explanation and in addition believed so-called albuminuric retinitis, in large part at least, to be a local edema of mechanical origin.

It is held by some authorities that an increase of cerebrospinal fluid is a necessary adjunct in the causation of choked disk. If this be so, it follows as a natural corollary that there must be a relatively diminished absorption. Whether this deduction be true or not has not been definitely demonstrated, though the experiments of Parker[13] seem to negative the proposition. That the choked disk of renal origin is brought about

12. Am. Jour. Med. Sc., cxxxvi, 484.
13. Jour. Am. Med. Assn., Oct., 7, 1916.

by increased intracranial pressure due to an increased secretion of cerebrospinal fluid is an attractive hypothesis. It is not difficult to build up a plausible theory. The cerebrospinal fluid is not a secretion in the true sense of the word. As demonstrated by Weed,[14], Dandy, and Blackfan, and others, it comes almost wholly from the choroid plexus. It is absorbed by the venous channels. A toxic agent might conceivably so injure both the delicate choroid plexus and the absorptive venous radicles that there would occur, on the one hand, a pouring out of increased cerebrospinal fluid, and, on the other hand, a lessened absorption.

The question might well be asked, Why does not choked disk then occur more frequently in nephritis? That is difficult to answer. In reply it might be said that renal choked disk is of serious prognostic significance. It indicates the end. The tissues of the brain, with the rest of the bodily tissues, can tolerate a considerable degree of slow poisoning up to a certain point. Renal choked disk occurs most frequently in the relatively young and before the tissues have accustomed themselves to a chronic toxemia. Cardiovascular complications often suddenly cut short life. Further one cannot positively say that choked disk does not occur in nephritis more frequently than reported, for the reason that, it being a terminal symptom, the ophthalmologist is not consulted. And again, it is possible for papilledema to be present with good central vision, the patient therefore giving no heed to his ocular condition.

Whether there can exist a true papilledema in nephritis without inflammatory phenomena in the nerve is difficult or impossible to say. It is my belief, and it is shown by microphotographs, that both conditions are found simultaneously in the nerve head. There may exist, however, a true optic neuritis without any choking. This is the usual finding in the vast majority of cases. The point I desire to make is that as regards pathogenesis the conditions are not interdependent, the neuritis being due directly to the renal toxemia; whereas, the choking results from increased intracranial tension and therefore indirectly the consequence of renal poisoning.

It may be extremely difficult, and even clinically impossible, to differentiate between an optic neuritis and a beginning choked disk, but that affords no reason for confusing the

14. Jour. Med. Research, xxxi, 1914.

genesis of the two conditions. The writer has never felt able, with absolute certainty, to clinically diagnosticate renal from other choked disk solely from the ophthalmoscopic findings. This point is settled by physical and urinary examinations. In general, however, it may be said that in tumor choked disk the storm center is the papilla, and the rest of the fundus may in large degree conform to the normal, though there are exceptions to be noted; whereas, in renal choked disk, while the papilla is extremely angry looking, the general fundus participates to a great extent.

In patients with renal choked disk whose skulls have been decompressed, the note is usually made that the cerebrospinal fluid was apparently under marked tension. Operation has availed little in these cases. It cannot hope to save life. At best it might prolong suffering; but it seems generally to have hastened the end. These unfortunate individuals should be spared operative interference.

Case Report.—C. A., aged 45 years, came to me Feb. 6, 1915. He had been having difficulty in near work for a year. He had never worn glasses. Sight had been getting progressively worse of late. Glasses for near work were desired. V = 20/75 R. E. and L. E. A preliminary test with lenses did not improve vision. Pupils were small and were dilated with euphthalmin for a fundus examination. The ophthalmoscope revealed a choked disk in both eyes, and a retinitis having the typical appearance of one of renal origin. Numerous scattered hemorrhages were present, some fresh, but the larger ones old and absorbing. The hemorrhage involved the perimacular region. Blood pressure = 225 mm. Hg systolic. Swelling of disks = 6 D. + R. E. and L. E. The patient has been treated for gastritis for a year with no benefit. There had been no previous ocular trouble. Patient had lost 50 pounds in weight in the last year. There had been no vomiting in the last few months, but some dizziness. Headaches infrequent. Field made, showing a marked concentric contraction in both eyes, more so in the left. Patient was put in the Barnes Hospital medical service, but was later transferred to the surgical service. February 23, Dr. Sachs did a right subtemporal decompression under local anesthesia. Previous to this, other fields were made showing a further loss of form field and a total loss of his color fields. No benefit generally or with regard to vision followed the operation. There was a slight subsidence of the swollen nerve heads following the decompression. The patient gradually failed, had several convulsions, thought to be uremic, and finally died April 5, 1915. Blood pressure varied from 186 (under nitrites) to 270. The following operative note was made by Dr. Sachs:

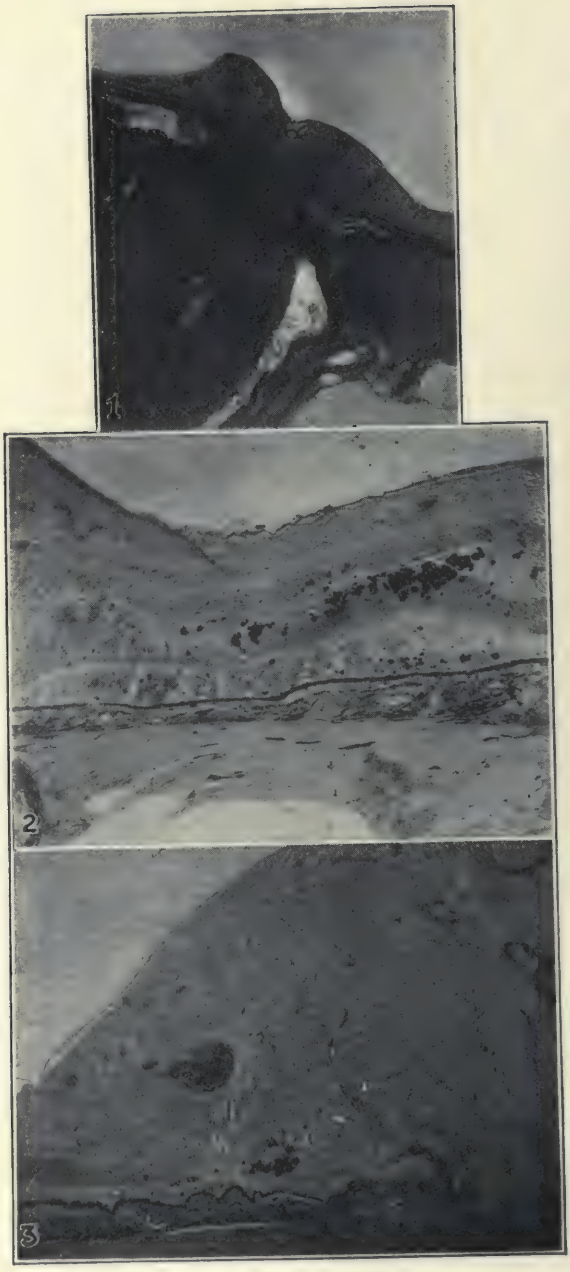


Figure 1 shows a section of the entire disk, figure 2 illustrates fat in the macula as stained with osmic acid; figure 3 shows a higher magnification of the disk, and depicts well the hyperplasia of glial tissue.

"Cortex looks very edematous and grayish, something like a wet brain. A great deal of fluid escaped. Brain tension plus, so that the brain bulged through incision." The clinical diagnoses were: Chronic diffuse nephritis, general arteriosclerosis, uremia, renal retinitis and choked disk. The postmortem diagnosis was chronic nephritis. Examination of the brain revealed many small sclerotic plaques of the basilar and vertebral arteries and a slight hernia cerebri. Otherwise it appeared normal. Immediately after death the eyes were obtained for microscopic examination, the preparation, sectioning and staining being done in the laboratories of the Washington University Medical School.

GENERAL PATHOLOGY IN BRIEF

In choked disk the space between the membranes surrounding the optic nerve is usually widened. Kampherstein found this to be so in 65 per cent of his examinations. Unless this has existed to a considerable degree and examination is made soon after death, it may easily be missed or obscured by postmortem changes.

Edema of the nerve trunk is said by many to be a prominent symptom of choked disk; whereas others, notably Paton and Holmes, have strongly denied this. They found no edema posterior to the entrance of the retinal vessels and no inflammatory changes in the optic nerve sheath.

Intracranial inflammatory processes do not in themselves cause papilledema. Infective cerebrospinal fluid, even a purulent fluid, may surround the optic nerve without causing an inflammation of the nerve or a papilledema. Often when the conditions are most favorable for the development of a neuritis it may not occur.

The anterior fibers of the lamina cribrosa are widely separated and arched forward. The posterior fibers may retain more or less their normal position. The nerve fibers are widely separated by fluid. The physiologic cup is filled in by the displacement of the fibers and by the edema.

The adjacent retina is displaced and thrown into folds. Separation of the membrana limitans occurs in numerous areas. These spaces occasioned by the separation of the limitans are often crossed by fine fibers. The internal limiting membrane is said to be made up of the spread out feet of Mueller's fibers, and it is these which cross the spaces. Paton and Holmes regarded these spaces as pathological and credited them with having to do with the formation of the white radiating macular lines occurring in renal retinitis. They are

no doubt produced by edema. Many are seen near the disk where the edema and its effects would be expected to be most pronounced. The vascular changes are chiefly a venous and capillary congestion.

It is unnecessary to enter into a minute description of the pathology of choked disk, which has been carefully done by Paton and Holmes and others, or of renal neuroretinitis, which Parsons has thoroughly described.

Dr. Alt went over the specimens of the case under consideration and has kindly furnished me with the following report:

"The most important histologic changes in the eyes of this patient were found in and around the optic nerve entrance, as was to be expected from the history. The papilla is considerably enlarged, not only in height, but also in breadth. Even in the hardened specimen it protrudes more than 1 mm. above the normal level. Microscopically this swelling is due to infiltration and edema. The infiltration, especially of the septa, is visible in the optic nerve as far back as it reaches in the specimens and is there more pronounced than in the papilla. The swelling of the papilla is chiefly due to edema, by which the nerve fibers and their septa are separated. There are a great many blood vessels visible in this edematous tissue. These are, to a large extent, the distended normal blood vessels; but I am inclined to think that many of them are newly formed. There were no hemorrhages in the swollen papilla in any of the sections which I examined.

"By the increase in breadth of the papilla, the beginning of the retina is naturally shoved for some distance outward over the choroid and, to a certain extent, detached from it. This detachment close to the papilla is due to a considerable amount of hyperplasia of glia tissue, farther away to a sanguinolent, partly fibrinous exudation between the two membranes, evidently the remnant of a large hemorrhage.

"While hemorrhages in the area of the papilla are wanting, characteristic ones are found in large numbers in the nuclear layers of the retina, and also a few in the nerve fiber layer. There is furthermore one large patch of varicose sclerosed nerve fibers in the nerve fiber layer on the nasal side of the papilla. This localized swelling is of more than double the thickness of the neighboring nerve fiber layer. The choroid also shows very marked changes. Due to some round cell infiltration around and in the neighborhood of the papilla and

to the hyperemia of its blood vessels, it is throughout thicker than normal. Almost all of the larger arteries show well developed endarteritic and periarteritic changes. In some the lumen seems to be obliterated.

"A further extraordinary fact is that there is an isolated tubercle in the choroid of one of these eyes, situated a little distance from the papilla. Here the overlying retina is also detached by a hemorrhage. This is the second time that I have seen the occurrence of a solitary choroidal tubercle in albuminuric neuroretitnitis.

Osmic acid staining showed the presence of fat in the base of the papilla and in the neighboring retina around the macula lutea and also in the exudates and hemorrhagic remnants lying between the choroid and retina.

"In spite of the considerable swelling of the papilla and surroundings in this case, the usual folding of the retina, and especially of its outer layers, is almost altogether wanting. The fact that a decompression operation did not remove the swelling of the papilla, shows that in this case the swelling and edema were not due alone to an increased intracranial pressure, but to inflammatory changes and new formation of tissue."

This case represents a typical instance of renal choked disk. The fact that the intense edema did not rapidly disappear following decompression has its analogy in certain cases of tumor papilledema. It is to be borne in mind also that in this case the choking was of long standing and intense, affording every opportunity for secondary inflammatory changes. Of interest is the microscopic appearance, showing a minimum of cellular infiltration of the papilla with marked edema and a pronounced cellular activity in the body of the nerve.

DISCUSSION

Dr. Marcus Feingold, New Orleans: I should like to place on record the following observations: A woman about 60 years old was brought into the Touro Infirmary about a year ago in an unconscious condition. The internist diagnosed uremic coma and she remained in this condition for several days. When again conscious she was totally blind, with pupils widely dilated. At this stage I examined the patient and found to my surprise, instead of the expected albuminuric retinitis, undoubted pure bilateral papilledema (choked disk) without any change in the retina and vessels. Elevation of the disk was about 5 to 6 D. In consultation with the internist and surgeon I urged decompression at once. On account of the bad condition of the patient the decompression was postponed for the time being and withdrawal of the spinal fluid substi-

tuted for it. This was done daily for three days, but no change in the eye took place. Then subtemporal decompression was done under local anesthesia, and two days later the patient began vaguely to see light. Gradually vision returned, and now the patient has sufficient vision to move around and even to see large objects.

Dr. George W. Mackenzie, Philadelphia: Dr. Jackson referred to the paper of Dr. Hardy. Parsons, Coats, and others have observed that in occlusion of the central artery it was possible to have also occlusion of the vein, because of endophlebitis, etc. Dr. Keiper's case was not a parallel of the one I am reporting. Furthermore, it was a case of occlusion of the central artery and not of a branch artery. As to whether the two conditions existed in my case, a study of the paintings should convince anyone. I did not try massage, because in a few cases reported it has done damage. I watched the eye carefully because of the possibility of secondary glaucoma following occlusion of the vein with hemorrhages.

Dr. William F. Hardy, St. Louis, Mo. (closing discussion): Unfortunately Dr. Lamb did not see my abstract or paper previous to its reading, and was, therefore, at a great disadvantage in discussing it. This was due to no fault on the part of either of us. I wish to take this opportunity of drawing the attention of the society to the evident injustice of designating a member for opening discussion on a paper without having put in his hands a copy of the paper to be discussed at least two weeks in advance of the meeting. I was not aware that Dr. Lamb was to open the discussion until the day before I left for Memphis. Dr. Lamb was evidently mistaken as to some of my conclusions. I am much gratified to have the support of Dr. Suker, and agree with everything he has said. His distinction between optic neuritis and choked disk was expressed more clearly than I have done it. The difference in appearance microscopically between optic neuritis and choked disk is nicely brought out by a number of lantern slides which I have, but which unfortunately cannot be shown because of lack of facilities in this room.

PERMANENT OCCLUSION OF THE SUPERIOR BRANCHES OF THE CENTRAL ARTERY—PROBABLY DUE TO THROMBOSIS OF THE CENTRAL VEIN OF THE RETINA.

GEORGE W. MACKENZIE, M.D.
PHILADELPHIA, PA.

The case that I am about to report is no less interesting and rare than it was confusing to me when first seen eight days after its onset. At that time, I was in doubt as to whether the case was one of thrombosis of the central vein, or one of occlusion of the superior branches of the central artery, for the fundus presented simultaneously the typical findings of each of these conditions. After the signs of thrombosis of the central vein had cleared sufficiently to permit of further study, there was unmistakable evidence of occlusion of the superior branches of the central artery.

The case was referred by the family physician, Dr. W. H. Phillips, of Cape May, and was first seen by me Dec. 1, 1914.

CASE 1.—The patient, Mrs. R. C. H., aged 59, robust looking but not obese, stated that she had enjoyed excellent health until ten days previous, when she contracted a severe cold in the right side of her head, and a day or two following noticed a slight pain in the eye with cloudiness of vision. So far as the patient can recall, the failure of vision came rather suddenly. She attributed the failing vision to the cold in the head. The cold has since improved, but while it lasted there was considerable discharge from the right side of the nose.

The urine was examined by her physician and found to be negative. She reported that her blood pressure was 180 mm.

Several days ago she suffered with slight headache in the back of the head, but has none today. She denied having tenderness about the frontal region or in the eye. The patient thought the vision was less blurred than it had been for several days. She said she was somewhat subject to bronchitis, but otherwise enjoyed good health. Her breathing through the nose was good at all times.

Previous to the recent cold she had no trouble with the eyes, nor had they ever been inflamed. One year ago she had a dizzy attack during which she felt as though everything was going around; however, it did not make her "sick at the stomach." She has not had a dizzy attack since and said she had no trouble with her ears.

External examination of the eyes showed the palpebral fissures to be normal, lid edges normal, ocular movements normal, ocular and palpebral conjunctiva slightly congested, but without secretion. Pupils were round, of equal size, reacted normally to light, accommodation and convergence.

Oblique Illumination. — Cornea brilliant and transparent in both eyes. Anterior chamber normal depth in both eyes. Irides blue and of normal texture. Pupils black, and no apparent opacities in the lens.

Ophthalmoscopic Examination.—The fundus of the right eye by the direct method gave the following findings:

(a) Blurring of the disk edge, except for the temporal quadrant, which was sharptly defined.

(b) Upper half of the disk showed a translucent bluish-white swelling, the lower margin of which was sharply defined and corresponded to the upper margin of the physiologic cup were it visible. The remaining outlines were limited by the upper temporal artery on the temporal side and the upper nasal artery on the nasal side. This swelling appeared to be very pronounced, 5 to 6 diopters, and pendulous. The most prominent part of the swelling, immediately above the lower margin, showed a distinct high light reflex which the artist has ably reproduced. A similar swelling on the surface of the skin would be promptly recognized as a blister about ready to rupture spontaneously.

(c) The edema which was so pronounced on the disk gradually diminished toward the periphery of the fundus. It was still visible three disk diameters above the disk.

(d) The edema even away from the disk was circumscribed by the two superior arteries as on the disk.

(e) Extending from the disk temporalward to the periphery was a large irregular elliptical area where the fundus was distinctly light in color. I find it difficult to describe the color exactly. It was more like a pale salmon color. However, it was decidedly different from the bluish-white color of the edema referred to previously. I accept the large pale salmon-colored area to be one of anemia due to cutting off of the blood supply. There is less of the anemia below than above the macula.

(f) In the anemic area of the retina the smaller vessels, both arteries and veins, were decidedly more conspicuous than in a normal retina.

(g) The macula showed slightly pink with the usual high light in the fovea, quite the same as in the normal eye.

(h) One of the most striking features was the radiating, narrow, streaked-like hemorrhage which covered almost the entire retina. These hemorrhages invaded even the anemic area in part. A study of illustration "A" will give a clearer idea than I am able to picture in words. A few of irregular shape hemorrhages were to be seen toward the upper nasal periphery, but are not shown in the painting.

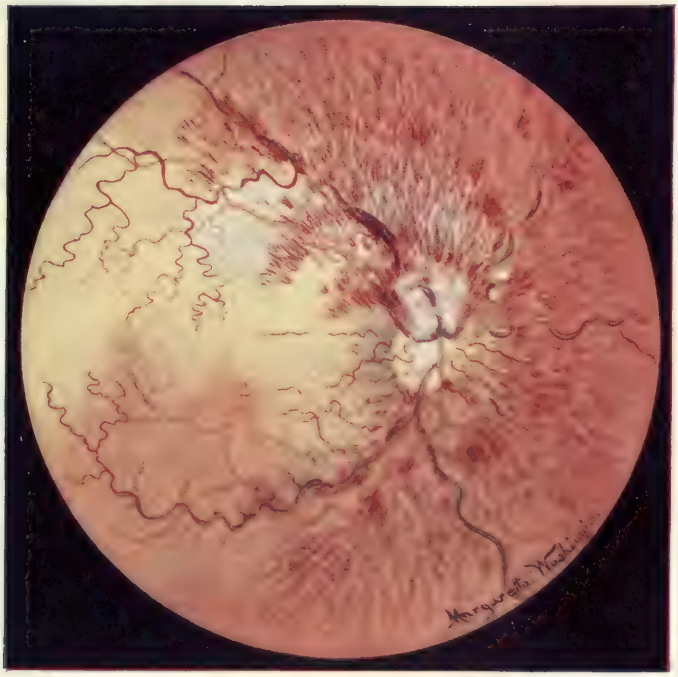
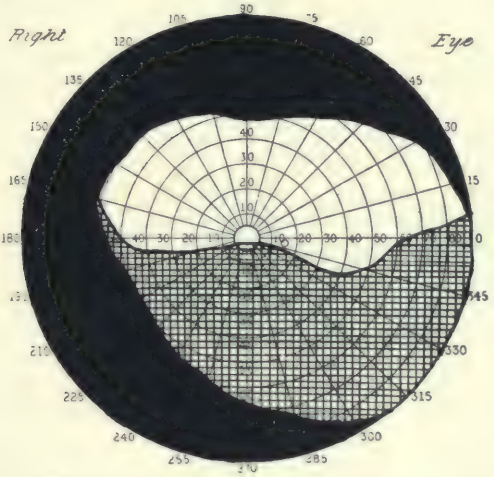


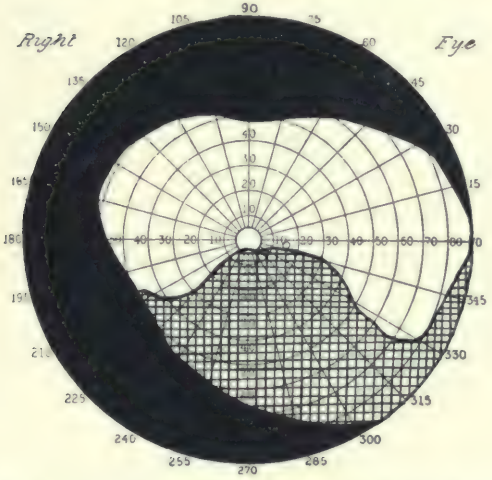
Plate I is a painting of the fundus of the right eye made December 5, 1914, four days after the patient's first visit or about 12 days after she first noticed obscuration of vision.

Plate II is a painting of the fundus of the right eye made March 28, 1915, about 16 weeks after the onset of her first symptoms of eye trouble.

(i) All the veins appeared to be dilated and more tortuous than normal, or as compared with those in the fellow eye. The most pronounced engorgement appeared in the upper temporal vein which was hidden in places by both the edema and the hemorrhages.



Field of Vision O D taken Dec. 18, 1914.



Field of Vision O D taken Nov. 18, 1915. This second field shows some improvement in lower half over that of the year before.

(j) The arteries generally appear to be of normal size in the painting, although observations made four days prior to the painting showed that the superior temporal artery was perceptibly narrowed just as it leaves the disk, while the column of blood within seemed to be dark and broken in places. The painting shows this portion of the artery somewhat obscured by the edema.

Ophthalmoscopic examination of the left eye gave normal fundus findings. Vision with present correction: R. E. with + 1.50 D. sphere equals 6/9 scant. The patient said she saw only the upper half of the letters, and fills out the rest with her imagination. She had quite as much, if not more, difficulty with the larger than with the smaller letters. Vision of the left eye with + 1.75 D. sphere equalled 6/6 full.

The visual field of the right eye was taken Dec. 18, 1914, and is herewith presented as first field. A later field taken Nov. 18, 1915, is presented as second field.

Blood pressure was taken and found to be 160 mm., systolic. Dr. Phillips reported subsequently that he found the blood pressure continued at about this same figure for several months.

Heart examination resulted in negative findings, nor had the patient ever presented symptoms or signs suggesting cardiovascular changes. Pulse was 70 and regular.

A second urinary examination was negative.

Intraocular tension measured with Schiötz tonometer showed R. E. to be 18 Hg; L. E., 23 Hg. Repeated examination for several weeks showed the same disparity in the pressure of the two eyes; that is, a lower pressure in the affected than in the unaffected eye.

Examination of the nose showed evidence of a recent cold in the right side, but no positive evidence of any sinus affection.

The patient was treated as an ambulatory one, with instructions to do no work and allow others to wait on her needs. Aspirin was prescribed in 15 grain doses for its diaphoretic effect. Red iodid of mercury, 1/100 grain, was applied every three hours and high frequency to the eye through the closed lid. Massage was not tried, believing it was too late, and from fear of doing the eye more harm than good.

The patient's eye, as well as her general condition, was observed critically for several months at close intervals early and at wider intervals later. She is still under observation and makes bimonthly calls at present, and is prepared to report promptly should any fresh disturbances of vision occur in either eye.

On Dec. 3, 1914, two days after the first visit, I fancied the edema on the disk was less pronounced; otherwise there was no apparent change until Dec. 12, 1914, when the patient reported that her vision was considerably improved, and with the test card she read 6/6 scant with the right eye. The edema was less marked on the disk and the veins on the disk seemed to be narrower than in the periphery. It was also noted that the veins in the periphery of the fundus still appeared to be distended. Otherwise the findings were the same.

Dec. 15, 1914, the edema on the disk had subsided sufficiently to permit a good view of the vessels, the cup and the lamina cribrosa. Relative size of arteries to veins off the disk is one to two instead of two to three. Striated hemorrhages were less apparent, were irregular in size, and extended far toward the lower periphery. These were quite black. Seven minutes high frequency to right eye was followed by an appreciable clouding of the vitreous, which I shall not attempt to explain at this time. At all events, the cloudiness prevented my seeing the lamina cribrosa, which was quite clear immediately before using the high frequency.

No appreciable change was noted in the fundus until December 21, when it was observed that central vision was quite normal, 6/6 full. Intra-ocular tension the same as on former occasions. The edema was decidedly less evident, so too was the anemia. The hemorrhages were clearing up fast. The superior veins began to show a narrower caliber, both on and away from the disk. The remaining hemorrhages appeared to lie near and parallel to the veins.

No further changes were noted until the week between Dec. 28, 1914, and Jan. 5, 1915, when it was observed that the radially running hemorrhages had almost disappeared. However, there were still some large, dark hemorrhagic areas of irregular shape low down and toward the periphery, which I am convinced were not there during the first week or two. The patient spoke of occasional floating opacities, which I was unable to discover in the vitreous.

By Jan. 23, 1915, all evidence of radially striped hemorrhages had disappeared; however, a few irregularly formed clots were visible in the lower periphery.

On February 16 both arteries and veins in the upper half of the fundus were smaller than in the lower half; the reduction in size of arteries was especially noticeable, and more so on and near the disk than farther away.

These same changes were noted on repeated occasions between February 16 and April 15.

On May 14 no hemorrhagic remains were visible. Disk was sharp in outline, upper half being quite pale, more so than is represented by the artist in painting B. This pallor, which was distinctly atrophic, had since become progressively more pronounced. The superior arteries were so small and pale that they were even more difficult to trace than they were in the illustration. There was no evidence of anemic pallor in the

region about the macula as was noted earlier in the case. Central vision is normal, 6/6 full. The field of vision resembles very much that of the one taken earlier; however, there has been an increase toward the peripheral lower nasal and peripheral lower temporal field, but no increase directly downward.

In summing up the most recent opthalmoscopic findings I can best do so by referring to illustration B, and adding slight modifications noted in the record but not by the artist. I did not wait sufficiently long for these later developments, being prompted by a natural fear that by waiting I might lose altogether the opportunity of securing a late picture.

The findings on Nov. 18, 1915, were as follows:

(a) Disk was sharply defined and quite white in upper half not noted in the painting.

(b) There was an increase in size of the cup, which is distinctly atrophic in upper half extending almost to the margin of the disk not noted in the painting.

(c) Lamina cribrosa was considerably more distinct than in the fellow (normal) eye.

(d) Veins in upper half of fundus were smaller in size than in lower half and especially on the disk. This difference is even more pronounced than when the artist made the second painting.

(e) Arteries in upper half of fundus were decidedly smaller than in the lower half. On the disk they were barely discernible. The diminution in size is less pronounced the farther we get away from the disk.

(f) About 1½ disk diameters away from the disk, along the course of the superior temporal artery, was to be seen a tuft of snow-white exudate. It appears to ligate the vessel, so to speak, at this point, for the vessel presents an entirely different appearance on the two sides of this tuft.

(g) The artery from the tuft of exudate just mentioned to the disk is so collapsed that it is barely visible. Even its walls seem to have shared in the atrophic process, for the white adventitious coat is less visible than shown in the painting. Toward the periphery from the tuft the vessel appears red, but like a thin and twisted tape. An artery running directly superiorly and the superior nasal artery appeared collapsed. They were pale near the disk, and pinker toward the periphery.

(h) The general color of the fundus was normal, as was also that of the macular region.

(i) Here and there in the upper half might be seen a few small scattered areas of degeneration.

In comparing the two pictures taken at different times and considering the history and progress of events up to this time, and in view of the present-day accepted teachings on the sub-

ject, I am compelled to accept the view that when first seen the patient had a double condition in the right eye, to wit: thrombosis of the central vein of the retina and occlusion of the superior branches of the central artery.

The symptoms and signs of occlusion of the central artery, so far as I have been able to collect them from the textbooks on ophtholmology, as as follows:

(1) *Sudden and complete blindness* in the case of embolism of the central artery without pain or prodromal symptoms. The entire field is affected in case the trunk is involved, but only a portion of the field in case it is a branch that is obstructed. Central vision may be maintained in those cases in which there exists a cilio-retinal artery of Zinn supplying the macular region (all authorities).

When the occlusion is due to a thrombus of the central artery, the patient is prone to suffer from temporary attacks of blindness both in the affected and the fellow eye, occasional headaches, dizziness and faintness (Priestley Smith).

After the first few days, there is frequently a slight improvement in sight, owing to partial restoration of the circulation from anastomoses of small vessels situated around the entrance of the optic nerve; but the improvement is very slight (Posey, Parsons). A case of sudden restoration of vision by anastomosis was observed by Lawson.

There appears to be considerable evidence in favor of the occurrence of a collateral circulation when the main artery is blocked (Parsons). This must be conceded to be rare.

(2) *Extreme arterial anemia* of the retina (Fuchs) is noticed immediately after the occurrence of embolism. I might say at this juncture that authors express themselves variously in referring to the pale color of the retina that follows promptly after the establishment of an embolus. Some refer to it as anemia, others edema and still others describe it in such an indefinite manner that the reader is left to guess what the author means. For instance, Fuchs says: "If immediately afterward an examination is made with the ophthalmoscope, the signs of an extreme *arterial anemia* of the retina are found (Fig. 251, Fig. 256 B, A and B). Within a short time, often within a few hours, the retina, as it dies, loses its transparency. It becomes of an opaque milky white, most markedly so in the vicinity of the papilla and of the fovea centralis." Nowhere does Fuchs mention edema, or anything that could be accepted as synonymous, in connection with obstruction of the central artery. On the other hand, de Schweinitz remarks that "An *opacity in the retina* develops in the form of a grayish-white, fog-like edema, etc." Elsewhere he says: "The author has seen the retinal edema and cherry red form in the macula within twenty minutes after the obstruction occurred." Referring to the pallor of the retina nowhere does

de Schweinitz mention the word anemia or its equivalent, but always refers to it as edema.

Ball in one place says: "If examined immediately after the accident, the ophthalmoscope will show complete absence of blood (another way of expressing the term anemia) from a part of or the whole retina, depending upon the location of the plug." On the same page (495), he says: "In a few hours the retina shows opacity due to edema." Page 495 presents also Figure 326, showing a section of the retina in embolism of the central artery (after Marple). A study of this section shows clearly an edema in the nerve fiber layer. Parsons (p. 1268) offers still another explanation of the cause of the pallor, concerning which he says: "It has been asserted with probability, on *a priori grounds,* that the retinal haze seen soon after the occurrence of the obstruction is not due to edema, as is frequently assumed, but to parenchymatous degeneration or coagulation necrosis of the inner layers of the retina supplied by the retinal artery." He qualifies this statement, however, by adding: "No case seems to have been examined early enough to prove this definitely, and edema has certainly been found later by several authors (Hirschberg, Nettleship, Nuel, Ridley, Manz)."

When excellent authors like these differ so radically in their interpretation of the retinal pallor, some of us are left to form from a careful study of our own cases a conjecture as to the cause or causes.

(3) At first the *arteries are thread-like* from more or less sudden cutting off of the blood-stream (Parsons). The main branches of the *artery* are thin, and can be followed only a short distance over the edge of the papilla into the retina and there is a diminution in the number of ramifications (de Schweinitz). The arteries are either completely lost to view or are much diminished in size.[1] The larger arteries are narrowed down to thin filaments; the smaller ones become invisible (Fuchs).

In what Parsons terms the second stage "there is some refilling from the establishment of a collateral circulation — we are looking with the ophthalmoscope at a small stream in more or less normal arteries." In the third stage, according to Parsons, there is fibrosis of the vessel wall — we are looking with the ophthalmoscope at a small stream in thick-walled arteries. There are several recorded instances (Schneller, Fischer, Wagenmann, Story) in which after the picture of complete obstruction there has been considerable recovery of sight. Lawson reported a case in which the arteries, which were thin on the disk, suddenly swelled out to their normal size as they crossed the edge, which points to the probability of an anastomosis. Coagulated blood may be found in the small arteries, particularly in the macular vessels (Ball).

(4) The *veins are contracted,* but may show irregular distention toward the periphery of the retina.[2] The veins are smaller

1. Amer. Encyc. of Ophthalm.
2. Amer. Encyc. of Ophthalm.

at the papilla, but somewhat increased in size toward the periphery (Swanzey and Verner). The vessels in the neighborhood of the optic nerve are indistinct in places, as if they were interrupted, while in the macular region the minute retinal vessels appear especially distinct (Haab). The veins are not markedly contracted except upon the papilla itself; the latter appears paler (Fuchs). The veins are contracted and very often present unequal distention, ampulliform broadening, alternate contractions and swellings and especially a contraction on the disk (de Schweinitz). There seems to be no contradiction in these findings among the different authorities that I have looked up.

The column of blood present in the vessels is sometimes broken up and moves about in an irregular manner; sometimes from one vein to another, sometimes in a reverse direction to the normal (Posey, Fuchs and others).

(5) Pallor *of the retina* about the disk and extending to the region about the macula. This was referred to above. In my own case the pallor in the macular region showed a tinge of yellow; at least it had a waxy color. It was positively not a pink-white, bluish-white or a snow-white color. The color did not suggest edema, for if it were due to edema of moderate degree it should show as a pinkish-white; if of pronounced degree, it should have appeared as a bluish-white or purplish-white with the element of white decidedly predominating, as I have seen before in cases of edema of the retina. Furthermore, I had the advantage of comparing the color of the macular pallor with the color of the known edema on the disk and partly beyond. The contrast was quite pronounced. The color did not suggest anemia as the cause of the pallor, but suggested rather the color of degeneration — the waxy degeneration as found in other organs. In the particular case that I am presenting of occlusion of the branches of the central artery, there was not the slightest suggestion of a cherry-red spot at the macula. The macula was a trifle less conspicuous than usual. The artist if anything has exaggerated it. It must be borne in mind that I did not see the case until at least eight days had passed. For this reason I have no intention of speculating on what the eye may have looked like prior to this time.

(6) *Cherry-red macula* is conceded by all authorities to be due to the choroid showing through the retina in this region, which anatomically is its thinnest portion. The color is made still more conspicuous by the contrast of the surrounding pallor of the retina. In rare cases of embolism the red spot may be absent. In the black races it has been observed as a black spot (Ball).

(7) *The smaller vessels, both arteries and veins, in the region about the macula show up more plainly than they do in the normal fundus.* This is due to the contrast of their red color with that of the pale background and not to an actual increase in their caliber. This explanation is conceded by all authorities.

(8) *The optic papilla is whitish or yellowish early* (Ball). Later on the optic disk is white, or of a dirty grayish color (Ball). The edges of the optic nerve are obscured by a thick white opacity of the retina which extends over the entire region of the posterior pole (Haab).

In Fuchs (p. 547) an embolism of the central artery developing eight days previous to date of observation in a woman affected with aortic aneurism, the whitish layer over the retina obscures the outlines of the papilla and the initial portion of the vessels arising from it. Atrophy of the disk follows late as a secondary change (Posey, Parsons). Atrophy of the retina and papilla supervene (Swanzey).

9. *Edema of the disk* is shown in Oeller's Atlas C, Table X, and in my own case still better. In referring to the disk, the descriptive text of Oeller says: "The disk is normal; the upper, inner and outer margins are sharply defined. The lower margin is completely covered by a whitish opacity which extends for some distance into the retina." The edema covering the lower margin of the disk extends with diminished intensity along and partly covers the lower temporal vessels.

The causes of obstruction of the central artery of the retina are: (1) Embolism secondary to valvular disease of the heart. This is at present considered to be a less frequent cause than formerly. Figure 147 in Orth's book on the pathology of the eye[2] is typical. One can see the round embolus in the artery behind the lamina cribrosa and an excellent illustration after Marple is to be seen on page 496, Ball's book.

(2) Thrombosis is perhaps conceded to be the most frequent cause; however, it should not be considered the primary factor, since it occurs only in conjunction with disease of the vessel-wall, namely, the next-mentioned condition.

(3) Endarteritis, proliferative endarteritis, is quite a constant pathologic finding met with in obstruction of the central artery or one of its branches.

(4) Arteriosclerosis is mentioned as a frequent factor. It need not be general, for many cases showing vascular changes in the eye do not show them at the wrist or even in the brain. Furthermore, the condition has been found in the vessels of the retina by pathologic study when they were not suspected by ophthalmoscopic examination, prior to enucleation.

(5) Aneurism of the carotid artery has been found a factor, but this has been rare.

---

2. Emboli der A. Centralis (after Schweigger).

(6 Ligation of all the carotids of one side is mentioned by Siegrist.[3]

(7) Spasm of the retinal arteries as described by Raynaud has also been observed in quinin amaurosis.

Cardiovascular diseases, kidney diseases, chorea, syphilis, and less frequently other systemic diseases act as contributory factors. In some cases no ascertainable cause has been found.

For other details of etiology and pathology the reader is referred to the works of Parson, Coats, and Orth.

The average textbook description of the symptoms and signs of occlusion of the central vein is even more meager than that of occlusion of the central artery. Fuchs devotes but a half page of text to the subject, while many others devote even less. The earliest and perhaps as accurate pictures of the fundi in obstruction of the central vein as can be found are given in Jaeger's Atlas and described by him as retinitis hemorrhagica, a term which has since been used by most authors as synonymous with thrombosis of the central vein. All authorities agree that the tendency to obstruction of the central vein is favored by age, cardiovascular sclerosis, and occupations which call for frequent stooping. It is almost always unilateral. (Parsons).

From a study of the literature upon the subject, I am led to include among the symptoms and signs of occlusion of the vein the following:

(1) *Diminution of vision* which tends toward eventually total blindness. In some isolated cases vision has been known to return. Coats' case, mentioned by Parsons, is that of a young lady in whom vision rose to 6/6 after the clearing up of the hemorrhages. Parsons further states that "When it is found that loss of vision is usually sudden, or at least not very gradual, and that there are usually not any prodromal symptoms, it might be concluded that the occurrence of a thrombosis and not of a very gradually built-up thickening of the vein wall would most probably be the immediate cause of the loss of vision."

(2) *Engorgement and tortuosity of the veins* is mentioned by all authorities. According to Orth, quoting from Leber, the veins were engorged in one case to two or three times the natural size and unevenly filled, and of dark brown color. In referring to single veins of the retina, Posey expresses his

3. Die Gefahren der Ligatur der grossen Halsschlagadern.

opinion thus: "At the same time the veins in the region are enormously distended, tortuous and dark in color, etc." It appears from a study of the literature and paintings that the distention is even more marked in cases of occlusion of a branch than of a trunk of the central vein. The amount of distention in the case of branch thrombosis is relatively easy to estimate, since we have more or less normal neighboring branches with which to gage dimensions.

In reference to the tortuosity of the veins, Parsons best describes the picture when he says: "The enormously swollen and tortuous veins at one place buried in exudation, at another thrusting forth a knuckle, etc." A study of the various illustrations in textbooks, atlases, and in my own case bears out this description.

(3) *Relatively thin arteries* is conceded by all authorities, except perhaps Haab, who, in referring to Figure 33b, says in the description that the arteries are not so much dilatated. However, a close study of the figure shows the arteries slightly narrowed, contradicting his description on the opposite page: "The arteries seem to be so attenuated as to be scarcely discoverable." There is a question in my mind whether the arteries are as much diminished in size as they would at first appear. Because of the enormous distention of the veins, the arteries no doubt appear to be thinner than in the normal fundus where the veins are seen in their natural size.

Furthermore, in Oeller's Atlas C, Table XII. (thrombosis venae temporalis superioris), it will be noted that the superior temporal artery is normal in size compared with the other branches. A study of Haab's Figure 33a (thrombosis of superior temporal vein), shows the corresponding artery to be normal in size compared to the other branches. The same holds true in Figure 2, Plate VII., opposite page 30, in Swanzy's handbook; also in Figure 191, thrombosis of a retinal vein, in de Schweinitz' "Handbook of Ophthalmic Practice;" also in Figure 832, thrombosis of a retinal vein, from a case of W. T. Lister in Parsons' work, "Pathology of the Eye." In the last mentioned figure the artery is more or less obscured by the hemorrhages into the retina. I am inclined, therefore, to believe that the diminution in size of the arteries, referred to by various authors, is more apparent than real.

On the other hand, pathologic changes in the vessel wall, such as sclerosis and endarteritis, that come with age, may so

alter the thickness, in some cases really, in other cases apparently, as to mislead us at times. Oeller in referring to C, Table XIII., says: "All of the larger arteries, even those running downward (normal portion of the retina) present very striking reflections."

(4) *Hemorrhage into the retina* is a constant finding in obstruction of the vein, and is generally recognized as one of the first prominent features. There are two distinct types of hemorrhage. The predominating type is the fine striated hemorrage in the nerve fiber layer which extends in a radial direction from the papilla. In the macular region they are relatively few as compared to the remaining portions of the retina, for the reason that the nerve fiber layer becomes less evident as we approach the macula. Every atlas and every textbook that shows the fundus in a recent case of thrombosis of the central vein, shows these typical hemorrhages in countless numbers. Haab in his Figure 33b shows the elliptical arrangement of the hemorrhages in the nerve fiber layer surrounding and a slight distance away from the macula. Ordinarily the hemorrhages in the nerve fiber layer so parallel the nerve fibers that individual nerve fibers seem to stand out upon a carmine or dark red background. Occasionally these radially running hemorrhages coalesce to more or less solid flame-like blotches. They maintain, however, feather-like edges which prove their character and location.

Oeller in referring to C, Table XI (thrombosis venae centralis retinae), speaks of a peculiar splitting of some of the blood patches below the grayish-black spot, suggests the formation of folds, possibly in the limitans interna. The same appearance was present in my own case a few days before Miss Washington made the first painting, and at the time I recorded the finding as one suggesting folds. It was not reproduced by the artist, for it had quite disappeared at the time when she examined the fundus. The second type of hemorrhages is that of irregularly shaped coagula which appear in the periphery and at a later time than the first mentioned. they are more solid looking, and according to most authorities occur in the deeper layers of the retina. Haab refers to the hemorrhages in the following words: "Circular hemorrhages occupy the deeper layers." In my own case they were found somewhat late in the equatorial region of the fundus and were very late in clearing up compared with the first-mentioned type.

Fuchs alludes to the recurrence of hemorrhages: "These keep recurring again and again and with their recurrence the sight which from the start was greatly reduced is at length utterly destroyed."

Ball claims that vision may be completely lost in thrombosis by repeated hemorrhages.

Hemorrhages into the retina are usually absent in occlusion of the central artery, and in one case of occlusion of a branch artery observed by Knapp, where hemorrhages did occur, it was no doubt due to occlusion of the corresponding vein which was engorged. Haab suggested that in Knapp's case the thrombus in the vein was the primary lesion and the obstruction in the artery was secondary to it.

(5) *Changes in the disk* are mentioned by only the minority of the authors. The majority simply pass them over without mention, perhaps because they do not find the changes characteristic or important enough to deserve mention. Haab observed that the optic nerve was indistinct, somewhat reddened and edematous, and contained a few stripe-like hemorrhages. De Schweinitz says: "There may be complete obscuration of the disk, which is hidden by infiltrated sheet-like hemorrhages which extend widely over the fundus." Bali claims that the margins of the optic disk are blurred and indistinct, but the disk itself is usually not much swollen.

The above description agrees quite generally with that offered by other authors for the early disk findings in case of thrombosis of the central vein.

The late disk changes are conceded by those who refer to them to be those of secondary atrophy. The ophthalmoscope shows atrophy of the optic nerve, with extreme attenuation of the blood vessels; but in this connection he is referring to a case of Knapp's in which there was a thrombosis consequent upon inflammation of the retrobulbar tissues, the result of erysipelas.

Posey mentions atrophy of the optic nerve following thrombosis of the vein, secondary to orbital disease, cellulitis, erysipelas, or disease of the cavernous sinus.

Swanzy refers to the optic papilla, which at first is swollen and congested, but after a time becomes pale and undergoes atrophy.

It is reasonably doubtful if the optic nerve undergoes an atrophic change as a result of occlusion of the vein except in

the cases mentioned by Fuchs and Posey. I have failed to find any reliable reference to atrophy in cases free from orbital disease. Parsons, who devotes twenty-six pages to the pathology of thrombosis of central vein, several times more space than any other author, does not refer to optic nerve atrophy.

I am inclined to believe from what information can be gathered that the optic nerve plays very little part in the pathology of thrombosis of the vein. The obscuration of the disk is merely due to the hemorrhages and slight edema in the nerve fiber layer, as it emerges from the disk, producing an opaque film, which simply shuts off our view of the margin of the disk. On the other hand, in case of occlusion of the artery there follows an unmistakable atrophy of the nerve. The distinction between the late findings of the disk in these two conditions is made more to avoid confusion in otherwise doubtful cases.

Edema is referred to by a few authors. Haab refers to slight cloudiness near the disk as due to hemorrhage and probably to edema. Wood refers to a serous effusion into the retina. Posey speaks of the affected part of the retina as being generally edematous and may contain spots or areas of exudation. Ball states that the veins may show on their convexity a light streak, while the concavity is hidden in the edematous retina.

Judging by the manner in which the authorities have ignored it and the slight and indefinite manner of referring to it by the few who do speak of it, we may safely conclude that edema is a relatively unimportant manifestation in this condition. If present, it is more or less obscured by the more evident hemorrhages. On the contrary, edema is quite in evidence in cases of occlusion of the artery, especially if the occlusion is of a branch, when the edema may be shown to best advantage on and a slight distance away from the disk (one or two disk diameters).

White foci of degeneration in the retina are mentioned by some authors, but not by others. None, however, attempt a description of them or explanation of their origin. De Schweinitz does not mention them; but Figure 191 shows these areas to exist. Haab refers to them as white foci of degeneration in describing Figure 33a; they are absent in Figure 33b. Fuchs does not refer to them in his text; but they are seen in his Figure 252 and are there referred to as having a brilliant white

hue in the center of hemorrhages. Posey refers to them as "spots and areas of exudation." Ball does not mention them in his text; but one fairly large area is seen in Figure 1, Plate XVII. Swanzy neither mentions them in the text, nor are they seen in his Figure 2, Plate VII. In Oeller's Atlas C, Table XI, they are not shown and not referred to in the description; C, Table XIII, thrombosis of the superior temporal branch of vein, shows many small round white dots near macula, to which he refers in the description as follows: "Between the bifurcation of a macular vein there is a group of small round white spots of fatty luster." Parsons does not mention or describe these areas of so-called degeneration in his twenty-six pages devoted to the pathology of thrombus of the central vein of the retina, very likely because he never examined them microscopically. In his Figure 830, "Vascular Degeneration" (of retina), he refers to the white spots of degeneration.

In the study of the case I am reporting, a few scattered areas are seen. From what I am able to sum up from the literature they are white—brilliant white according to Fuchs; fatty luster according to Oeller. The latter is rather an exceptional view. I gather further that they appear late in a case and are probably secondary to the hemorrhages, and appear in central areas of the larger hemorrhages. Since no one else has suggested it, I am left free to offer the opinion that they are the natural sequences of organization of a clot of blood and as such should appear as brilliant white scars, and a close study of the fundus of my own case further bears out this opinion. In short, they are in a pathological sense areas of organization and not those of degeneration.

For the various pathological changes which have been put forward in explanation of the clinical picture of thrombosis of the central vein, we have, according to Coats:

(1) A thrombosis in the central vein in eleven out of twenty-two cases.

(2) Occlusion of the central vein by proliferation of the intima, but without thrombosis (possibly in Michel's case).

(3) Multiple thrombi in the retinal veins, but without a thrombus in the central vein (Alt, Ischrey).

(4) Multiple emboli or perhaps thrombi in the retinal arteries (Wagenmann, Case 1). Stolting seems to regard his case as of the same sort. In Freidenburg's and Meyerhof's cases there were thrombi (or coagula) in both arteries and veins.

(5) Changes in the retinal vessels (hyaline thickening, endarteritis, endophlebitis) sometimes amounting to occlusion, but not the result of either thrombosis or embolism (Wagenmann, Case 888; Deutschmann, Case, 2; Renner).

(6) Hemorrhage into the substance of the optic nerve (Deutschmann, Case 1).

For a definite description of the pathology of obstruction of the central vein of the retina and the bearing that this condition has to glaucoma, the reader is referred to the excellent treatises by Parsons, Coats, and Orth.

The combination of obstruction of the central vein with that of obstruction of the central artery has been mentioned by several authors.

The first case of the kind was that of Knapp, which has been freely quoted by other authors since. In Knapp's case, the ophthalmoscopic appearances seemed to indicate that there had occurred an embolism of a single arterial branch in the retina, in which the corresponding vein was engorged and accompanied by hemorrhages. Haab's explanation of Knapp's case is that a thrombus in the vein was the primary lesion, and as a result of this obstruction an endarteritis had been set up in the artery, narrowing its lumen.

In Coats' Case 3, thrombosis of the central vein of the retina, pathologically examined, he found a little cellular plug in the artery, of which he says: "There can, I think, be little doubt that it is an organized thrombus, and that the thrombus in the artery was secondary to the interference with the blood flow caused by the oclusion of the vein." Orth quotes a case cited by Leber in which there was an obstruction in a branch vein combined with a narrowing of the accompanying artery to the extent that the artery was thread-like on the disk.

Swanzy claims that in "thrombosis of the retinal vein (hemorrhagic retinitis) a secondary thrombosis may occur in the central artery and then a white cloudiness of the retina will appear in addition to the signs of venous thrombosis."

Ball in referring to the diagnosis of embolism of the central artery (p. 497), says: "Another lesion which obscures the diagnosis is thrombosis of the central vein at a point where it can pass on and occlude the adjacent artery."

Bankwitz reports a case in which he found by pathological examination small aneurism of the artery in front of lamina cribrosa, supposed to have obstructed the vein and caused

thrombosis of it. Retinal arteries were much thickened and in places obliterated. Veins were also thick, some containing thrombi.

Würdemann cites a case in which a clot was found in the artery from 1 mm. behind the lamina cribrosa backward for 5 mm., and a small clot in the vein not completely occluding lumen.

Myerhof reported a case of a male, aged 50, whose left eye was removed for glaucoma, secondary to hemorrhagic retinitis, and whose pathologic examination showed thrombi of both arteries and veins.

Gonin tells of a case of a woman, aged 52, who suffered from mitral insufficiency and albuminuria. A postmortem pathologic examination of the left eye revealed a granular mass penetrated by endothelial cells in central artery; farther forward a canalized thrombus in the vein, no doubt of long duration.

Other cases might be cited in which there has been found the co-existence of occlusion of the vein with occlusion of the artery; but they could serve only the purpose of corroborating the experience of those already quoted.

Returning to my own case, in view of the history, the ophthalmoscopic findings, later developments, and in the absence of pathologic study, I am able to arrive at but one conclusion, and that is the coexistence of occlusion of the superior branches of the central artery with occlusion of the accompanying veins. My reasons for reaching this conclusion are as follows:

(1) There positively existed an occlusion of the veins as evidenced by the typical hemorrhages throughout the fundus, the enormously dilatated and tortuous veins, the subsequent areas of so-called degeneration.

(2) The above findings do not belong to but are foreign to occlusion of the artery, which is admitted by all authors to be a fact.

(3) There positively existed an occlusion of the artery as revealed by the marked edema of the optic nerve, from the physiologic cup in an upward direction to a disk diameter and more; by the characteristic pallor of that part of the fundus corresponding to area ordinarily supplied by the superior branches and especially toward the macular region; by the distinct narrowing of the upper anterior branches as compared to the other branches; by the still further dwindling in size of these vessels two or three months later, and by the subsequent atrophy of the upper half of the disk.

(4) The findings just cited are *not* those of occlusion of the veins, nor have they ever been accepted as such by any authority.

(5) The co-existence of occlusion of the central artery with occluson of the central vein has been reported by trustworthy authorities.

(6) Where this co-existence has been found it happened more often in the branches than in the main trunk of the vessels.

Lastly, the fact that the patient was totally blind in the lower half of her field with preservation of her upper half, indicates that the upper half of the retina was affected with a graver lesion than the lower half. Furthermore, since the greater portion of the retina, including the lower half, showed the characteristic findings of occlusion of the vein (see painting), we may conclude that the defect in vision for the lower half of her field was not due to the occlusion in the vein. Finally, since there was obstruction in the upper arterial branches and not in the lower ones, it follows that this occlusion in the arteries was the especial cause of her loss of vision in the lower field and that the occlusion of the veins played little or no part in her visual defect. The evidence then clearly points to the fact that the patient suffered from obstruction of the superior arteries at the time when the first field was taken, and in all probability, judging from her history, even earlier, if not from the first moment when she observed cloudiness of vision.

BIBLIOGRAPHY

Priestley Smith: Ophth. Rev., 1884, **3.**
Posey: Diseases of the Eye.
Parsons: Pathology of the Eye, 1908, **4.**
Lawson: Tr. Ophth. Soc., U. Kingdom, 1898, **18.**
Fuchs: Textbook on Ophthalmology, 1913.
De Schweinitz: Diseases of the Eye, 1915.
American Encyclopedia of Ophthalmology, 1915, **6.**
Ball: Modern Ophthalmology, 1913.
Swanzy: Handbook of Diseases of the Eye and Their Treatment.
Haab: Atlas of Ophthalmoscopy and Ophthalmoscopic Diagnosis, 1901.
Haab: Bericht über die 26. Versamml. d. ophth. Gesellsch., Heidelberg, p. 160.
Harms: Arch. of Ophth., 1905, **66.**
Orth: Lehrbuch der speciellen pathologischen Anatomie, Berlin, 1903.
Siegrist: Die Gefahren der Ligatur der grossen Halsschlagadern, Leipzig, 1900.
Oeller: Atlas of Ophthalmoscopy, 1896-1899.
Coats: Thrombosis of the Central Vein of the Retina, Royal Lond. Hosp. Rep., 1904, **16,** part 1.
Coats: Obstruction of the Central Artery of the Retina, Royal Lond. Hosp. Rep., 1905, **16,** part 3.
Knapp: Arch. f. Augenh., 1869, **1,** 29.
v. Michel: Arch. Ophth., 1878, **24,**, 2; Deutsch. Arch. f. klin. Med., **22,** 439; also, Ztschr. f. Augenh., 1899, **2.**
Meyerhof: Ztschr. f. Augenh., 1900, part 4, 676.
Wagenmann: Graefe's Archiv, **40,** p. 221; also, Arch. Ophth., **38,** 213.
Würdemann and Deutschmann: Beitr. z. Augenh., 1898, **29,** 100.
Bankwitz: Arch. Ophth., 1898, **45,** 384.
Wood: A System of Ophthalmic Operations, 1911, **2.**
Gonin: Arch. d'Ophth., 1903, p. 219.

DISCUSSION

Dr. Edward Jackson, Denver, Colo.: I think Dr. Mackenzie is fully justified in the diagnosis of thrombosis of the central vein of the retina. In looking at the plates it might be thought the arteries present greater changes as a final result than the veins. But I think this is not unusual. Harms pointed out some years ago that almost invariably when one set of vessels was obstructed the other became obstructed in a very short time; that we do not have a thrombosis or embolism of the arteries without obstruction of the veins, and vice versa. The two go together. I have seen one case that I believe was an incomplete thrombosis of the vein. The patient recovered entirely. This was seventeen years ago. I see the man ocasionally, and I think he has good vision. The vein was never entirely lost, but there were clouds and marked changes of the retinal veins. The arteries at no time showed a marked change. That was the only case I ever saw in which there was obstruction in one set of vessels, in which I did not think there was obstruction in the other.

I have never seen a case just like that of Dr. Feingold. There is incomplete coloboma of the iris in the sense that it did not extend to the periphery of the iris. But on transillumination there was a coloboma of the retinal pigmental layer, so that the red pupil extended to the periphery of the iris. I have seen a good many cases in which I thought the retinal layer of the iris was so thin that the light sifted through all parts. One of these was after a cataract operation done by another operator. It was very marked and there was no evidence that the patient had in early life suffered from disturbance of vision; but after the lens was removed there was marked disturbance. I have been surprised many times to find an iris that transmitted light through all parts of it, when I had not expected from the external appearance of the iris that such a condition was present.

Dr. Robert S. Lamb, Washington, D.C.: As I understand it, he is limiting himself to papilledema in conjunction with an acute condition of nephritis. I prefer to consider it choroido-retinal, as it is almost never retinal alone. I prefer to think of the kidney as an excreting organ, and it does not seem possible it alone has the toxic ability to produce this picture. I think you invariably find the forerunning condition of a noncompensating heart. You cannot explain by mechanical means the inability of the blood to return. You find orbital strictures and the turgescence of the nose and throat, which will interfere to some degree with the return of any of the lymph, and the constant pressure of that against this spongy wall will prevent the blood coming through, which will be bound to result in a hemorrhage if it is kept up. I have gone over in the hospital a number of times a number of cases in the wards to make an attempt to diagnose by the ophthalmoscope alone acute nephritis with a high blood pressure, marked nephritis with definite arteriosclerosis and the fundus picture of albuminuria associated with a definitely leaking heart. The picture of papilledema with hemorrhage must of necessity be of relatively long standing. I do not mean two or three weeks or more, but it depends on the intensity of the process. It might be a few days, or a week, before the hemorrhage stage would be reached. I believe just as firmly that if we should make an ophthalmoscopic examination, we should find we had a beginning papilledema and a beginning hemorrhage associated with albuminuria and high blood pressure. I cannot go into the subject from its various points of view as I should like, but I want to draw out a few points to give Dr. Harris a chance, in the light of his more recent investigation.

Dr. George Keiper, Lafayette, Ind.: Ten years ago, at the St. Claire meeting of this Society, I reported a case of obstruction of the superior temporal branch of the central artery of the retina, and for fuller details I refer you to the publication of the transactions for that year. The interesting thing was that we could see not only the point of obstruction of the vessel, but also the characteristics of the retina beyond. I speak of this at this time because the essayist has not mentioned the way to treat these cases. Prior to that time, Dr. H. W. Würdemann had made the statement that the only thing that would do good was deep massage. Force the eye back in the orbit and let it go. Repeat several times. Uveitis followed in a couple of years, which cleared up, and we found complete atrophy had taken place in that spot and patches of choroiditis were found throughout the fundus. The patient was apparently a healthy young woman, accustomed to dressmaking. I put her on specific treatment, which cleared the trouble up and left these atrophic spots. If we expect to get any results, we must resort to deep massage of the eye, and inhalations of amyl nitrate may be used. I have seen one case of complete closure of the central vein of the retina. The edema was very marked and with extensive hemorrhages. This cleared up, but left the eye blind. I shall report this case later.

TUBERCULOSIS OF THE RETINAL VESSELS

FRANK R. SPENCER, A.B., M.D.
Instructor in Oto-Laryngology, University of Colorado
BOULDER, COLO.

Tuberculosis of the eye is as protean in its manifestations as tuberculosis of the other organs of the body; for instance, this disease may affect the lids, conjunctiva, cornea, sclera, iris, ciliary body, choroid, retina, and perhaps even the lens. If inflammation of the lens existed or were possible, the lens would doubtless respond in this way to the toxins produced by the tubercle bacilli. In fact, lenticular opacities may be, and very likely are, due to autogenous or bacterial toxins circulating in the blood and lymph. Until this is better understood, we may not successfully prevent these opacities or cure them after they have formed. However, it is the object of this paper to deal solely with tuberculosis of the retina and retinal vessels.

The earliest intimation I can find in the literature that hemorrhage from the retinal vessels may be due to tuberculosis is in an article by Noll[1] in 1908. He states that hemorrhages into the vitreous may be due to tuberculosis of the choroid and ciliary body. Arnold Knapp[2] is second in an article entitled "Form of Retinal Tuberculosis." In speaking of the juvenile vascular disease and exudative retinitis, he intimates that tuberculous disease of the retina is the underlying cause. He reported two cases with distinctly retinal lesions, vitreous haze, and superficial disturbance of the choroid. The tuberculin test gave general and local reactions, but he does not say whether or not there was a focal reaction. Under treatment with tuberculin, the lesions at first extended, then gradually disappeared, but left serious impairment of central vision.

Agricola and Thies[3] examined an eye that had suffered from serious tuberculous keratitis and iritis, and that had healed under tuberculin treatment, but left the eye blind. Later cerebral symptoms arose and the eye was enucleated. There was chronic tuberculosis of the ciliary body, with but slight involve-

1. Noll: Ztschr. f. Augenh., 1908, p. 548.
2. Knapp: Arch. f. Augenh., **75**, 259; Amer. Ophth. Soc., **13**, 486.
3. Agricola and Thies: Klin. Monatsbl. f. Augenh., 1913, Jan., 20.

ment of the choroid, and the retina seemed to have become infected through the vitreous. Tissue like that of retinitis proliferans was found near the disk. There were tubercles in the retina, chiefly perivascular.

In this particular case it seems quite evident that the retinal disease was secondary to that of the cornea and iris, so that the report of Agricola and Thies need not necessarily be included in my paper. On the other hand, the severe and extensive involvement of the cornea and iris doubtless prevented early ophthalmoscopic examinations, so that, even had the retinal lesions been primary, these could not have been diagnosed prior to the enucleation.

While we probably should not regard all cases of juvenile vascular disease or periphlebitis as tuberculous in character, the tubercle bacillus is responsible for the majority of the cases. Stock[4] has reported on the anatomic examination of one eye of a patient dying from miliary tuberculosis. He had suffered obscure symptoms before admission to the hospital. Eight days after admission, there were retinal hemorrhages in both eyes, which were ascribed to general sepsis. On the day of his death, three miliary tubercles were found in the left choroid. The histologic examination showed hemorrhages in the nerve fiber and ganglion cell layers of the retina. No bacilli were found in the retina, but the hemorrhage appeared to be due to general tuberculous infection.

Cords[5] has reported two cases, and collected six others, all of which were probably tuberculosis of the retina. His two cases improved under tuberculin treatment.

Igersheimer[6] saw three cases of retinal tuberculosis, and two of his cases had pulmonary tuberculosis, just as my second case had.

Gilbert[7] has reported three cases of periphlebitis, which he thinks show that tuberculous disease of the anterior segment of the uveal tract may cause phlebitis of both the uveal vessels and the central retinal vein. In each case the tuberculin reaction was positive.

Fleischer[8] reports a case of retinal periphlebitis occurring in a man of 36, who suffered from pulmonary tuberculosis. The age of this man is nearly the same as that of my first case.

---

4. Stock: Klin. Monatsbl. f. Augenh., **51,** 75.
5. Cords: Ztschr. f. Augenh., **26,** 441.
6. Igersheimer: Graefe's Arch. f. Ophth., **82,** 215.
7. Gilbert: Thirty-ninth Ophthalmologic Congress, Heidelberg, p. 47.
8. Fleischer: Klin. Monatsbl. f. Augenh., **52,** 769.

One eye showed periphlebitis with extensive hemorrhages. These were gradually absorbed with return of vision. A year later there was iritis, followed by secondary glaucoma, for which the eye was enucleated. Some of the veins were found filled with tuberculous debris.

In one of Harms'[9] cases of periphlebitis and retinal hemorrhages there was gradual improvement after a few weeks, and, although some lesions remained, vision rose to normal. This patient seemed to be in fair general health, but suffered from tachycardia on slight exertion and gave a weak positive general tuberculin reaction.

Oloff[10] reports two cases of tuberculosis of the retinal veins. The patients were young men, sailors and robust. They gave distinct general reactions to tuberculin. Oloff finds these cases are not benefited by tuberculin treatment, but often do well under general dietetic and hygienic measures.

In one of the five cases of retinitis with massive exudates reported by Friedenwald[11] there was a marked tuberculin reaction.

In a paper on retinal tuberculosis by Otori[12] two cases seen clinically are reported, and also experiments upon rabbits concerning ocular tuberculosis. In the first patient the left eye was enucleated on account of a hemorrhagic inflammation involving the iris and cornea. With the microscope, the retina was found to be extensively destroyed, the nasal portion being entirely necrosed, and a number of typical tubercles being present. Tubercle bacilli were demonstrated. This is the first case of primary retinal tuberculosis in which tubercle bacilli have been found in the tissues. A modified staining method was used for the microscopic sections. They were stained for from two to four hours with hot carbolfuchsin solution; then placed for a short time in a 0.5 per cent. hydrochloric acid-alcohol with some tincture of iodin, and washed in distilled water; then differentiated in a solution of picric acid with indigo-carmen (saturated aqueous solution of picric acid 200 gm., indigocarmin 0.5 gm.). The staining process was completed with alcohol and carbolxylene. The tubercule bacilli were found only in parts of the retina which were relatively little changed. They were especially numerous in masses of

9. Harms: Graefe's Arch. f. Ophth., **87,** 457. Ophthalmology, **11,** 108. Ophth. Rev., **33,** 231.
10. Oloff: München. med. Wchnschr., **61,** 1103. Clin. Ophth., **20,** 661. Jour. Am. Med. Assn., **62,** 2054.
11. Friedenwald: Tr. Am. Ophth. Soc., **13,** 819.
12. Otori: Arch. f. Augenh., **79,** 44.

round cells along the veins of the retinal periphery, and could not be found at all in the uveal tract.

In the second case the left eye was enucleated on account of hemorrhagic glaucoma secondary to a severe plastic iritis. In this case the anterior half of the nasal side of the retina was necrosed. Tubercle bacilli were found in every part of the retina, and they were extraordinarily numerous in a large expanse of the equatorial region. The large colonies of bacilli in the retina were sharply limited by the pigment epithelium layer, no bacilli being found in the choroid. They occurred in a few sections in the posterior part of the ciliary body, and were numerous on the optic nerve head. As regards the first case, Otori believes that the bacilli had at first their seat in the optic nerve head, and from there invaded the other tissues. The second case he believes to have been one of primary retinal tuberculosis. He regards the secondary infiltrations in the uvea and also in the optic nerve trunk of both cases as in the nature of a toxic inflammation.

The experiments performed upon rabbits included: (1) inoculation into the common carotid artery; (2) injection into the veins of the ear; (3) inoculation into the common carotid artery with simultaneous division of the ciliary vessels; (4) inoculation of the common carotid artery with ligation of the common jugular vein of the same side; and, (5) inoculation of the common carotid artery with ligation of the common jugular vein. With none of these methods was a tuberculous localization of the disease obtained, although by some of them a tuberculous infection was produced in the uveal tract. From these experiments Otori concludes that the rarity of primary tuberculosis does not depend merely upon peculiarities in the retinal circulation, but rather upon a natural lack of disposition of the retina toward primary tuberculous infection. The clinical cases reported are to be considered as arising by way of a perivasculitis of the retinal veins, directly caused by migration of tubercle bacilli into the lymph channels of the retina.

Hemorrhage from the retinal vessels in young adults is almost always due to tuberculosis of the retinal vessels. Axenfeld and Stock[13] expressed this opinion six years ago. It is, perhaps, needless to state at this point that I am excluding all cases of trauma, early arteriosclerosis, syphilis, hemophilia, cardiovascular diseases in general, thrombosis or embolism of

13. Axenfeld and Stock: Klin. Monatsbl. f. Augenh., January, 1911.

the central artery or vein, nephritis, etc. One should always be on the alert for changes due to syphilis, and this disease should be excluded in all cases of retinal hemorrhage. In the past, too many cases of tuberculous disease have been regarded as syphilitic and treated with mercury and potassium iodid, when they should have received injections of tuberculin. Hemorrhage here is just as characteristic of tuberculosis as hemorrhage from the lungs in the pulmonary form of the disease. Knapp[14] has reported two cases in which hemorrhage from the retinal vessels into the vitreous was a very prominent symptom of the tuberculous disease. His two cases are as follows:

"Case 1.—The ophthalmoscope showed a swollen and inflamed optic disc. The swollen nerve head seemed displaced by an ill-defined chorioretinal area, up and in, surmounted by a small hemorrhage; up and out and next to a blood vessel, there was a superficial, round, white focus; a smaller, though similar, patch was seen below, a short distance from the disc. The macular region was occupied by a perfect star-like figure. The spokes were composed of glistening, silver interrupted lines. The diagnostic dose of 3 mg. O. T. produced two days thereafter many round, white areas about the disc and the larger retinal focus. He remarks that it is uncertain whether the tuberculin injection made these small foci ophthalmoscopically visible or whether they represented the dissemination of the general focus. He further states that rapid and continuous improvement, under specific tuberculin treatment, confirms the diagnosis of tuberculous lesion."

"Case 2.—In this case the ophthalmoscope showed white exudates, composed of round areas, which surround the course of the macular vein. This vessel was unevenly dilated and there were small hemorrhages on the exudate. Three mg. O. T. was followed by focal reaction and vitreous haze."

A chronic form of choroidal tuberccle, "Obsolescent tubercle," in strumous children, first described by Sydney Stephenson[15] and considered by him to be tuberculous, by use of tuberculin diagnostic test has been definitely determined to be tuberculous (T. Harrison Butler and Karl Koller of New York) The latter described the reaction to be an opacity of the vitreous; change of color of the tubercle, more reddish.

In fact, the hemorrhage into the vitreous if more marked, or subhyaloid if slight, with subsequent loss of vision, may be the first intimation the patient has of the disease. On the

14. Knapp: Arch. Ophth., **42**, 592.
15. Stephenson: Lancet, London, 1915.

other hand, the hemorrhages may recur several times, especially if only one eye is involved, before there is sufficient obscuration of vision to attract the patient's attention. This was undoubtedly true in my first case reported in this paper.

Fuchs[16] states, in the last English edition of his book, that "hemorrhagic retinitis is due, for the most part, to diseases of the retinal vessels; many of these cases are probably identical with those of thrombosis of the central vein, as described on page 549." However, he does not mention tuberculosis as a cause.

George E. de Schweinitz[17] mentions tuberculosis as one of the many causes of retinal hemorrhage and he refers to Dimmer's classification. Roemer[18] mentions the fact that tuberculosis may be present in a patient with retinal hemorrhages without being the cause.

One striking feature of the disease is the fact that such cases rarely show other evidences of tuberculosis, even after a careful and thorough physical examination by one or more competent internists and the patients often look the "picture of health." It is, therefore, necessary to make a tuberculin test, as Posey[19] has mentioned, before the diagnosis can be made. The second case reported in this paper is an exception to this rule. There may be deep seated tuberculous glands somewhere in the body, or one or more very small tuberculous foci deep in the lungs.

At least the intra-ocular tuberculosis is endogenous in its origin and is only one of the tuberculous foci within the body, as Gamble[20] has mentioned. In any event, these do not show, even in a very careful physical examination, but these isolated tubercles may rupture into a blood vessel and produce infection of the retinal vessels by metastasis. This, at least, seems to be at the present time, the most logical explanation. However, these patients do, in many instances, react to tuberculin and give not only a local and general reaction, but a focal reaction as well. A mild general or local reaction in adults, as is now well known, is of very little importance, because so many adults will react to tuberculin. For instance, in Vienna, 90 per cent. of the adult population will react to tuberculin.

---

16. Fuchs: Duane's fourth edition of Fuchs' textbook of Ophthalmology, p. 551.
17. De Schweinitz: Diseases of the Eye, Eighth edition, 452.
18. Roemer: Textbook of Ophth. (as translated by Foster), **3**, 740.
19. Posey: Ophth. Rec., 1916, January 25.
20. Gamble: Ophth. Rec., 1916, Feb., 55.

A positive history of tuberculosis is very difficult to obtain in health resorts, especially in Colorado, except in the last stages of the disease, as Jackson[21] has previously mentioned. For this, as well as for other reasons already mentioned, patients with recurrent hemorrhages from the retinal vessels should receive very thorough examinations from ophthalmologists, internists, etc., because so much depends on a proper diagnosis being made early.

Another symptom, which is just as important, is the vasculitis and perivasculitis with fibrosis about the vessels. The fibroid changes undoubtedly occur subsequent to the hemorrhages, although they go hand in hand in any given case, and both lead ultimately to the condition known as retinitis proliferans or proliferating retinitis.

Mayou[22] calls attention to the fact that the infection may gain entrance to the retina along the optic nerve sheath, and he reports a case to illustrate his point. The fundus presented an appearance not unlike albuminuric retinitis with exudates. Parsons[23] explains this by saying the "Transmission by the lymph stream is probably the commonest mode of infection, so that the sheaths suffer most frequently and worst."

Manz[24] has described, under the name of retinitis proliferans, an affection in which dense masses of connective tissue extend out from the retina into the vitreous and cover a portion of the fundus; in fact, even the papilla itself. Into these masses run new formed vessels from the retina. For a number of these cases it is probable that these masses of connective tissue have been preceded by hemorrhages which were poured out from the retina into the vitreous and afterward became organized. However, he does not suggest the possibility of tuberculosis.

Retinitis circinata, as described by Fuchs,[25] is doubtless only one form of an atypical type of proliferating retinitis and is probably due to tuberculosis of the retinal vessels. The fibroid changes are again characteristic of what we know concerning tuberculosis elsewhere in the body, i. e., in the lungs, larynx, etc., and represent Nature's efforts to arrest further progress of the disease. However beneficial these changes

---

21. Jackson: Ophth. Rec., 1916, Jan. 1. Amer. Ophth. Soc., **14**, 577. Ann. Ophth., **25**, 84, 177.
22. Mayou: Tr. Ophth. Soc. U. Kingdom, **34**, 180.
23. Parsons: Pathology of the Eye.
24. Manz: Duane's fourth edition of Fuchs' textbook of Ophthalmology, p. 552.
25. Fuchs: Duane's fourth edition of Fuchs' textbook of Ophthalmology, p. 554, l. 21.

may be in other organs, they are more or less destructive of vision when they occur in the eye.

Therefore, the therapeutic administration of tuberculin, proper diet, and an outdoor life will do much, in many of these cases, to save the remaining vision and will often restore some of that which has already been lost. Jackson[21] has undoubtedly saved the vision of two of his cases by this treatment. On the other hand, Black[26] has a patient who has not done well in spite of all his skilful treatment. This case is the exception, as there may be a mixed infection present.

New tuberculin (T. R.) should be administered in doses of 1/10,000 to 1/2,000 mg. It is best to begin with very small doses and gradually increase. The injections should not be repeated in less than two to four or even six weeks. We should give too little, as Parker[27] has mentioned, rather than too much. Also Gamble[20] has emphasized this. An exception to this is when tuberculin is given for diagnosis. Then it may be repeated every, three, four, or five days until a reaction is obtained. If old tuberculin (O.T.) is used 1/10 to ½ mg. may be injected at the same intervals.

However, tuberculin is like all other remdies; it will not prove suitable for all cases. Von Hippel,[28] out of his large experience has called our attention to the fact of the common relapses of the disease, following, or in the course of, tuberculin treatment: "Among seventy-five cases of tuberculosis of the iris and ciliary body, relapses occurred in fifteen and at intervals of from three months to three years after the end of first course of treatment. In 115 cases of tuberculosis of the cornea, there were twelve relapses. In eighteen cases of keratoiritis, three; in eight cases of conjunctivitis, two. No relapses in cases of sclera or choroid. The total number of relapses were thirty-two out of 243 patients. A long interval must elapse before one is justified in considering tuberculosis of the eye definitely cured."

In fact, many patients object seriously to the use of tuberculin, especially if they experience the slightest reaction, even locally. This was true with the first case in this paper. I agree with McCool[29] when he says "he would almost prefer to be the second oculist consulted rather than the first, aside

26. Black: Ann. Ophth., **25**, 397. Tr. Colo. Ophth. Soc., 1916, Apr. 15; Ophth. Rec., 1916, Sept.
27. Parker: Ophth. Rec., 1916, Jan., 21.
28. Von Hippel: Graefe's Arch. f. Ophth., **87**, 193.
29. McCool: Ophth. Rec., 1916, Jan., 8.

from the satisfaction of having made the diagnosis, because such patients often seek advice elsewhere." This unpleasant experience may often be avoided by injecting tuberculin in very small doses and at long intervals. This is very important to remember for the following reasons:

(1) Moderately severe or severe reactions can be avoided. In most injections there should be only a slight local reaction, if there must be any reaction at all, because if patients respond with a moderate or severe general reaction they are very likely to have new retinal hemorrhages, as evidence of the focal reaction, with loss of more vision. This is more likely with the first few injections than after the patient has been under treatment for a time.

(2) The psychologic effect of small doses is desirable, since patients are much more likely to continue this important line of treatment. Remember, too, that this treatment requires months, or even several years, but even this prolonged treatment is better than blindness.

The administration of tuberculin in the past has been left very largely to the internist, who usually has only a limited knowledge of fundus diseases. Much more progress in the treatment of the disease can be made if ophthalmologists will at least direct the treatment, as the fundus should be examined the second and third day following each injection of tuberculin, especially if there is any reaction. This is very necessary until the diagnosis is well established, but is, perhaps, not quite so necessary every time thereafter.

The fact that tubercle bacilli for very obvious reasons cannot be obtained for smears counts for nothing in the diagnosis. Collins[30] has reported eighteen cases in which the eye had been enucleated for uveal tuberculosis. In one of these cases tubercle bacilli were found, in another their presence was doubtful, and in four cases they were looked for and not found. In one case a successful inoculation was made in the eye of a rabbit. But in fifteen cases the diagnosis seems to have rested on the symptoms, clinical course, and pathologic histology. As Jackson[21, 22, 23] stated, it is experiences like this that have caused the staining and search of suspected tissues for bacilli to be ranked below inoculation tests, as a foundation for the diagnosis of tuberculosis. The diagnosis of tuberculosis of the retinal vessels, by the diagnostic use of

30. Collins: Ophthalmoscope, 1907, Jan., Feb. and Mar.

tuberculin, is more positive than the diagnosis of a syphilitic iritis, because a patient gives a positive Wassermann. However, we should not have undue faith in tuberculin, even in positive clinical tuberculosis, as this test cannot always be depended on. Von Hippel[31] reports a case in which the enucleated eye was found full of tuberculous masses; yet the general reaction to old tuberculin was only obtained from the sixth injection containing 5 mg.

Perhaps three cases to illustrate the ophthalmoscopic findings, subjective symptoms, diagnosis, etc., will be of interest. The first one is so typical, it is what the German and Austrian ophthalmologists call a school or textbook case:

Case 1.—Mr. A., aged 32, a farmer, whose weight was 190, looked well and felt well. He was first examined March 31, 1916. He gave a history of failing vision in his left eye, dating from

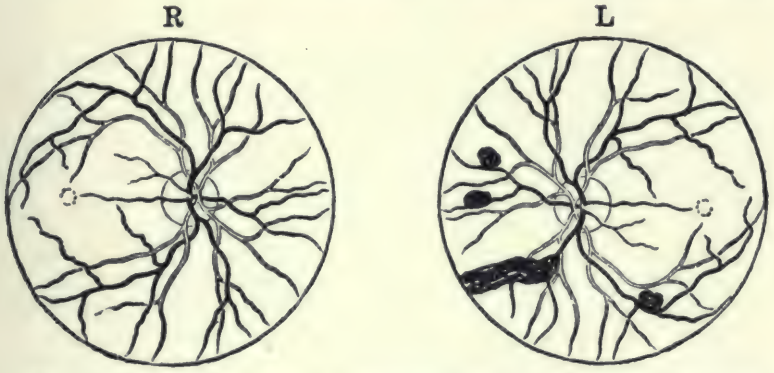


Fig. 1.—Round areas represent hemorrhages—long area represents proliferating retinitis.

Jan. 8, 1916. At first, when his vision began to fail, he saw a streak before his left eye, but, as the sight became worse, he scarcely distinguished even the streak. V. R. E. = 15/20— and J. No. 1 and V. L. E. = 2/100 and not even J. No. 11.

The anterior segment of each eye was negative and the right fundus was negative. However, examination of the left fundus revealed the following: Vitreous was very hazy; disk looked very hyperemic, as one would expect through a hazy vitreous, and there were many hemorrhages about the retinal vessels with areas of retinal proliferation. In this case, as in the other cases of retinal tuberculosis, the organization of the exudate, with the formation of fibrous tissue, as manifested by the proliferating retinitis, goes hand in hand with the hemorrhages. The disease in this patient must have lasted longer than the history indicated.

The hemorrhages were most numerous in the nasal, inferior nasal and inferior portions of the fundus. The largest streak of

retinitis proliferans was about one of the inferior nasal vessels. The accompanying illustration shows this better than any description can do (Fig. 1).

He denied venereal disease and the luetin test was negative. He gave a very decided local and general reaction to 1/10,000 mg. of old tuberculin and a slight, doubtful focal reaction. A Wassermann test was advised in spite of the negative luetin test, and, in case the former should be negative, as it probably would be, treatment with tuberculin. He refused to have the tuberculin used or the Wassermann test made, as he was prejudiced. The following is the report from his physician, Dr. Clay E. Giffin, to whom he was referred: "Chest and heart negative; abdomen negative; blood negative and blood pressure, 127 systolic by Tycos instrument. He gave a general, local and a slight focal reaction to an intracutaneous injection of old tuberculin (O. T.). Patient refused to return for subsequent tests because of a general sense of well being, which seemed to make further examination useless."

Case 2.—Mrs. B., aged 27, was a married woman and the mother of two children. She was the chief support for the family, as her husband had been blind several years following the explosion of a misspent shot in a mine. She was first examined Aug. 24, 1916. She stated that the sight failed suddenly in her left eye one week prior to this. She had V. R. E. 6/6— and J. No. 2. V. L. E. was reduced to light perception with good projection, but she was unable to recognize or count fingers. The anterior segment of each eye was negative and the right fundus was negative, but the examination of the left fundus revealed a massive hemorrhage in the vitreous, so that the details of the eye grounds could not be recognized. Upon close questioning, this patient admitted that she had bronchitis, active pulmonary tuberculosis and that she raised sputum each morning. A thorough examination by her family physician, who is a competent internist, revealed an active tuberculous lesion in the apex of the right lung. The report of Dr. O. M. Gilbert is as follows: "Aside from eye symptoms, patient complains of fever, weakness, cough and expectoration. She has two children 8 and 10 years old, but has had several induced miscarriages."

"Fall of 1912 had an abscess on the kidney and was ill for several months, then well until the fall of 1913, when she was 'nervous and run-down' and had a hacking cough. Never well since nor free from cough. Spring of 1915 had five pulmonary hemorrhages of a pint or more each. Ran a fever ever since and has lost thirteen pounds."

"S. P. Temperature 100.8; pulse 116; weight 124; blood pressure 110/70; dyspneic on exertion; coughing and expectorating freely. Occasional night sweats, no more hemorrhages."

"*Examination.* — Considerably emaciated, no edema, pallor, jaundice or pigmentation. Pupils and reflexes negative. Urine negative, blood, luetin negative. Sputum showed a great many tubercle bacilli, few streptococcus and staphylococci. Heart nega-

tive. Lungs: Right.—Consolidated in apex and infiltrated to third interspace, with probably small cavity behind the second rib. Numerous crackling and small bubbling râles throughout this region and posteriorly down to inferior angle of scapula. Left.—Consolidated in upper half with large cavity from first to third rib, infiltrated throughout the remainder except the lower margin laterally. Tuberculin tests not given."

Dr. Ross C. Whitman, professor of pathology, University of Colorado, reported the blood Wassermann negative.

Case 3.—Mr. C. was first examined Sept. 13, 1916, and gave the following history: Aged 20, and single. He stated that his left eye had been slightly inflamed for a few days previous and that objects looked smoky with this eye. He stated that he could see the outline of people, but could not recognize their faces. He thought the sight failed rather suddenly in his left eye a few days before. He denied having had tuberculosis or venereal diseases, but stated that he was quite nervous before coming to Colorado. V. R. E. 6/5— and J. No. 1—; V. L. E. 6/6 plus and minus and J. No. 2.

The right fundus was negative, but the examination of the left revealed fresh blood in the posterior portion of the vitreous chamber, so that it was impossible to see the details of the retina and disk very distinctly. The disk "looked red," as one would expect through a hazy vitreous. There was enough blood in the vitreous, so that I could not detect the vessel or vessels from which the hemorrhage had occurred. While I could see the disk reasonably well, the vision was much better than one would expect from the objective findings in the fundus.

The following report is from his family physician, Dr. O. M. Gilbert: "In addition to his eye symptoms he complains only of having had persistent colds quite frequently, occasional gastric distress shortly after meals, which disappeared upon quitting coffee; also having been run down during the past spring and summer. S. P. No cough. Temperature 99.2; pulse 104; weight 122."

"*Examination:* No edema, pallor, jaundice nor pigmentation. Fairly well nourished, but not muscular. Pupils, reflexes, gait and station normal. Heart and lungs negative. Abdomen negative, except for a deep tenderness over the appendix. Luetin test negative; urine and blood negative. Blood pressure 109 systolic, 70 diastolic."

"Tuberculin test:

September 14, O. T., 0.0001 mg., only slight reaction.

September 17, O. T., 0.1 mg., slight general but no local reaction.

September 25, O. T., 2 mg., slight general reaction, but no local reaction.

September 30, O. T., 10 mg., more marked general and local reaction.

Temperature 99.2, no focal in lung."

Dr. Ross C. Whitman reported the blood Wassermann negative.

After being given diagnostic doses of tuberculin, he gave only a very slight, rather doubtful, focal reaction, with a mild local and general reaction when the dose reached 10 mg. This failure to react to the smaller doses was probably due to the fact that a very small, isolated tuberculous foci in one lung, in a lymph gland, etc., is not large enough to produce decided local or general reaction. This is certainly true with a small tuberculous focus in the retina, as the general toxemia from such a small focus is practically nil.

I have been able to find in literature twenty-nine cases of retinal tuberculosis, which, with my three cases, makes a total of thirty-two. I could perhaps have found other cases in the lierature and among my own records, but I have purposely excluded all doubtful cases.

In conclusion let me call your attention to a most excellent symposium upon ocular tuberculosis by Jackson, McCool, Posey, and Gamble, read before the Pacific Coast Oto-Ophthalmological Society, June 17, 1915, and published in the *Ophthalmic Record* for January and February, 1916. I have quoted freely from this symposium and I believe every ophthalmologist should read the articles comprising it.

DISCUSSION

Dr. Edward Jackson, Denver: I think there are enough cases now on record to draw a rather distinct line between these retinal cases and those of choroidal disturbances. Choroidal tuberculosis is certainly not generally attended with hemorrhage. On the other hand, the retinal cases, which it has been suggested are secondary to lesions in the ciliary body and perhaps of the cornea and iris, will ordinarily be attended with retinal hemorrhage and at an early stage. In all the cases I have seen there has been vitreous opacity, whitish clouds entirely separate from the retina, supporting the idea that the retina is reached through the vitreous, secondary to diseases in the anterior segment of the eyeball. I think if a case follows one of these clinical lines of progress that we have the material now for a practical diagnosis, aside from the Wassermann reaction and therapeutic tests.

Cabot, reporting the results of diagnosis in some 3,000 cases coming to autopsy, found in a very large percentage of them no complete diagnosis had been arrived at before death. Diagnoses often cannot be made with positiveness in the first week or month, and we should adopt tentatively a probable diagnosis based on the clinical course. When we have material to do that with safety, we shall be less at the mercy of our knowledge with regard to specific tests. A case I reported eighteen months ago as one giving no evidence of active disease for several months, continued so through pregnancy and for three months after the child was born. Then the patient had a fresh retinal hemorrhage, and in the past nine months she has had three or four hemorrhages. She

had one course of tuberculin therapy and she improved, and has had no fresh hemorrhage for three or four months, but has some signs of failing general health, although no tuberculous lesions have been recognized in the lungs.

I was glad to hear in Dr. Spencer's paper about the length of time between the tuberculin injections. He put the minimum at two weeks. That is certainly as correct as those who advise giving it with a maximum interval of one week, and perhaps every two or three days. I think two weeks is perhaps not too long a minimum; and, while some of these doses seem very small, it certainly is safer to go slow in the administration of tuberculin. It is only the rare case that seems to indicate that they do better after a tuberculin focal reaction; and the slightest reaction we can recognize is probably the ideal reaction.

Dr. Joseph Lichtenberg, Kansas City: Referring to Dr. Spencer's paper, I wish to report a case corroborative of the observation that the patients are in good health and generally free from tuberculosis in other parts of the body.

A young male, aged 18, had failing vision in the right eye, the left eye being normal. Vision, fingers at four feet. A typical optic neuritis was much as seen with the ophthalmoscope. Wassermann was negative. Physical examination was negative; also the urinary examination. I gave an injection of O. T. and had a local and general reaction. He made a practically perfect recovery, with vision perfectly normal under tuberculin treatment.

Dr. John Green, Jr., St. Louis: I agree with Dr. Spencer that many of these individuals are in splendid general health. I recall in particular a man, with weight over 200 pounds, and with rosy cheeks, who had well marked tuberculosis of the iris, yet he gave a general and focal reaction to tuberculin. Under tuberculin therapy, his eye recovered. Choroidal tuberculosis is commonly regarded as luetic, and these patients are given mercury and iodids, with damaging results. After establishing the diagnosis, it is advisable to begin tuberculin treatment in conjunction with an internist, for one never knows how small a therapeutic dose may give rise to general reaction. On one occasion, in a case of corneal tuberculosis, without the cooperation of an internist, I injudiciously began with T. R. 1/10,000 mgm. There was a prompt rise in temperature, with the appearance of râles and cough. After an interval it was found that he could be started with 1/100,000 mgm., which, gradually increased, effected a cure. In the case of Dr. Woods' patient, who apparently was made worse by tuberculin injection, might not the mercury which was subsequently given have been the damaging agent?

Dr. Hiram Woods, Baltimore: The object of the paper was to call attention to two things: that the reaction might be dangerous and the use which therapeutically we may get along without. Dr. Spencer gave a case in which 3mg. were used for diagnostic purposes. The patients I have to deal with would run me out of town if I gave that dose. Dr. Green's statement would not hold with my case, because the patient got worse after she went home and took mercury. It was a definite damage following tuberculin.

Dr. F. R. Spencer, Boulder, Colo. (closing discussion): I do not believe I have anything to add to what has been stated, except to mention that in all of these cases the accessory sinuses were negative. In one case there was active pulmonary tuberculosis. A majority of these cases

do not give a clinical evidence of tuberculosis and in the first case the man cannot believe he has tuberculosis of the retinal vessels. The third patient has good health and one would not be inclined to think of tuberculosis of the retinal vessels. The question is to determine the dose, and I don't believe you can lay down any rule. The safest rule I know of is to begin with a small dose, and stop short of reaction, as Dr. Jackson says.

SOME PHASES OF THE DIAGNOSTIC AND THERAPEUTIC USES OF TUBERCULIN IN UVEITIS

ILLUSTRATIVE IN FOUR CASES

HIRAM WOODS, M.D.
BALTIMORE, MD.

I want to present the essential features of four carefully studied cases of uveitis, thought to be of tuberculous origin, three of which were treated with tuberculin. These cases seem to justify the study of three phases of this form of therapy:

(1) The possibility of permanently damaging vision by a focal reaction.

(2) Advisability of omitting a focal reaction and relying for diagnosis on a positive von Pirquet, or other diagnostic measures for tuberculosis, after elimination of other causes.

(3) Conditions indicating the use or avoidance of tuberculin.

CASE 1.—Permanent damage to vision following the diagnostic use of tuberculin.

Mrs. S., 31 years of age, mother of three children, consulted me on Aug. 2, 1912. The right eye was destroyed two years previously by central choroidoretinitis. Vision in the left eye had been poor for ten days. Her previous history was negative, except for convulsions before the birth of her second child. She had been subjected to repeated systemic examinations. No serologic tests had been made.

Examination showed: R. E.: Central and paracentral scotoma, foveal choroidoretinal atrophy with numerous spots of disseminated atrophy throughout the fundus.

L. E.: Central vision, 20/40, form and color fields normal. In the lower periphery, there were numerous pigmented atrophies, with minute grayish yellow deposits, certainly of recent date, near the temporal disk margin.

She was sent to the hospital for clinical study. Nothing abnormal was found except a high specific gravity in the urine (1.030) with indican in excess. A Wassermann on the blood serum was reported negative. Temperature taken every two hours for two days showed no variation. Then on August 4 an internist, associated in the case, gave without my knowledge, 0.5 mg. of tuberculin for diagnostic purposes. There was no temperature reaction; the sole observable result was focal. Five days after the use of tuberculin, she complained of diminished vision in the left eye. It was 20/100. The vitreous was more cloudy and between the fovea and disk there was a new choroidal

exudate. The patient went home after another week, unimproved. While there she took mercury under the direction of her physician. She returned to Baltimore after three months with vision the same as when she left, 20/100; a paracentral color scotoma, contracted form field and an atrophic spot near the fovea. A second blood serum Wassermann, made by another serologist, was reported "feebly positive." Because Mrs. S. had taken mercury during the three months spent at home, he believed that the diagnosis of syphilis should be accepted.

The points I want to emphasize are:

(1) Presence in each eye of atrophic areas, while the better eye showed in addition such signs of acute disease as fresh exudates and cloudy vitreous.

(2) The speedy occurrence of new trouble in the *acutely affected* eye after injection of tuberculin with permanent lessening of vision.

(3) The meaning of positive reaction in the second Wassermann test. In view of the first negative Wassermann the feebleness of the second and the decided tuberculous reaction, I am inclined to think the diagnosis of syphilis not proved.

This is a matter of some importance in view of a suggestion by Theobald Smith.[1] In a paper from which I quote later, he speaks of living organisms becoming pathogenic when introduced into a body infected with another virus. True, living organisms are not used in human beings; but the effect of various preparations of dead organisms is the same, only to a less degree. It is certainly conceivable that a vaccine which would react only slightly in a body not otherwise infected, might cause greater damage because of previous infection.

Case 2.—Rapid extension of choroidal exudate following diagnostic use of tuberculin.

Miss A. R., aged 19, a Jewess, public school teacher, consulted me Dec. 31, 1915, for floating spots and blurred vision in the right eye, of a fortnight's duration. Both eyes were myopic to nearly 5 diopters. With correcting glass, the right eye had 20/30, central vision, the left 20/20. A fine descemetitis could be made out in the right eye. After mydriasis, there was found slightly cloudy vitreous with a large choroidal exudate in the lower nasal field.

Examinations, including sinuses, teeth, blood serum Wassermann reaction, etc., showed nothing save a slight enlargement of the right lobe of the thyroid. The patient was sent to the hospital, and put to bed. The temperature, taken every two hours for two days, showed no fluctuations. Then on the advice of

1. Theobald Smith: An Attempt to Interpret Present Day Uses of Vaccines, Jour. Am. Med. Assn., **60**, 1591.

Dr. H. D. McCarty, who thought that such a dose could do no harm, I consented to a subcutaneous injection of 0.001 mg. tuberculin for diagnostic purposes.

Three days later, the temperature reached 100 degrees, and there was circumcorneal congestion; also, along the border of the choroidal deposit originally observed, except at its lower and outer margin, there was a deepening of the red color of the fundus. This was displaced in two days by a new grayish yellow exudate. Vision fell to 20/40(—). This focal reaction subsided after two weeks, when the patient was given a 0.00001 mg. O. T. as a therapeutic measure. During the four months following, Miss R. received twelve injections of tuberculin, the strength gradually increasing to 0.10 mg. She usually showed circumcorneal reddening, but no other reaction. Her central vision became normal in six weeks and has remained so ever since. A small incut found in the tempero-superior field in the beginning gradually cleared. She has had no treatment since June, 1916, and has remained perfectly well.

I have cited this case to show the possibility of a severe focal reaction from reasonably small doses of tuberculin. The rapid improvement may or may not have been due to this vaccine. I do not believe we can say positively, for the case was acute, and Nature might have done the same thing without our help — or interference — as the case may be.

Case 3.—One eye lost from uveitis; the other apparently saved by tuberculin.

T. B., colored, aged 22, a waiter, was first seen April 11, 1913. He was married and had one healthy child. He had lived in a house with two sisters who had died of tuberculosis. In the summer of 1912, his left eye was lost from uveitis. The globe was shrunken, the sclera thinned and the tension minus. The pupil was occluded and the lens opaque. He said he had light perception. So far as I could determine, mercurial treatment had been administered during the attack in the left eye.

The right eye showed steamy cornea, descemetitis, posterior synechiae with vitreous too turbid to admit of fundus examination. Vision was moving hand at two or three feet. He was sent to the Presbyterian Hospital and Dr. Gordon Wilson kindly examined him for me. Clinical examinations and Wassermann reaction on the blood serum were negative. From April 19 to 21 the temperature remained normal or below, the pulse was 90. A von Pirquet test was ordered, and the resident physician was entrusted to make it. Probably he overdid it. At any rate, in forty-eight hours there was violent arm reaction; the temperature rose to 101.6, the eye became more injected and painful. I am inclined to think there was an accidental subcutaneous test. We decided to limit treatment to atropin and dionin until the temperature reaction should cease and the inflammatory lesion should become nonprogressive. It was nearly a month, May 17,

before this was accomplished. The patient was then admitted to the Municipal Tuberculosis Hospital on Dr. Wilson's service. There was no vision except the moving hand. There were present deep circumcorneal injections, adherent pupil and muddy vitreous. He remained in the Municipal Hospital until Sept. 16 (four months), with the exception of one week in July. He received all together twenty-two injections of tuberculin, all after the first two being bouillon filtrate. The dates of administration, the size of the doses, and such notes as were made at the hospital, are appended.[2]

Evidently Dr. Wilson's purpose was gradually to increase the dose to a point just short of febrile or focal reaction. The man received no other general treatment, while locally atropin and dionin were continued.

He was discharged from the Municipal Hospital, Sept. 16, 1913, with normal central vision. He has been at work since and came to my office at my request on April 3 of this year. The lost left eye is unchanged. In the right there are a few adhesions along the lower pupillary border. An area of choroidal atrophy is seen in the upper and temporal fundus with numerous small pigment deposits elsewhere, especially around the fovea. He had 20/20 vision and seemed in good health.

I call special attention to the avoidance of tuberculin so long as the case was in an advancing stage.

Case 4.—Chronic disseminated choroiditis with repeated circumcorneal injection and scotomata; the former apparently controlled, the latter diminished by tuberculin.

Mrs. W. B. R., aged 45, consulted me first in July, 1908. The right eye had been useless for a year or more from disseminated choroiditis with central atrophy. Vision was: R. E. 20/200, L. E. 20/20. If there was any trouble in the left eye at that time, my notes do not indicate it. Mrs. R. presented the appearance of perfect health. She lived in the country, and outside of what she termed "chronic rheumatism" had never been ill. The one significant thing in her family history was that four brothers had died of tuberculosis. She had been examined repeatedly and nothing had been found. At that time (1908) I corrected only the presbyopia.

---

2. Case 3. Record of injections and doses with dates: May 28, 1913, 1/10,000 T. R. (T. rose from 96 to 98.4. No entry of focal reaction.) June 3, 1913, 1/100,000 B. F. No note of reaction, local or otherwise. June 11, 1913, 1/100,000 B. F. June 15, 1913, 1/5000 B. F. Elevation of T. from 97 to 99.2. For one week (until June 22) had oscillating T. Generally subnormal not exceeding 99. From 6 a. m. on June 22 to 12 noon on June 23, T. 97 to 99.2. June 23, 1913, 1/5000 B. F. June 28, 1913, 1/5000 B. F. July 2, 1913, 1/5000 B. F. July 5, 1913, 1/5000, T. elevated 1°. July 9, 1/3300, slight oscillation of T. all below normal. Home: July 16 to 24. July 26, 1913, 1/3300. Low variation in T. continued, from 96 to 98.6. Aug. 8, 1913, 1/2500. Aug. 12, 1913, 1/2500. Aug. 15, 1913, 1/2000 from then to Aug. 20 highest T. 98.2, lowest 95 (on two occasions). Aug. 20, 1913, 1/2000. Aug. 24, 1913, 1/2000. Aug. 27, 1913, 1/1888. Aug. 30, 1913, 1/1888. Sept. 2, 1913, 1/1500. Sept. 10, 1913, 1/1500. Sept. 12, 1913, 1/1200. Sept. 14, 1913, 1/1000 B. F. Sept. 16, 1913, dismissed. T. usually from 95 to 98, 99.2 on only two occasions and followed injections of 1/3300 B. F.

I did not see Mrs. R. again until May, 1913, five years later. She then came on account of recurrent redness in both eyes. There was episcleral and circumcorneal injection in her left eye. This (the good eye) showed numerous patches of disseminated choroiditis, chiefly in the upper and inner fundus. Figure 1 shows her left field at this time. Note the large scotomata. Central vision was 20/20, in spite of a fine descemetitis. She was being treated by her physician with sodium salicylate in the belief that rheumatism was causing the eye congestion. This treatment was continued for five weeks, or to July 2, without appreciable change.

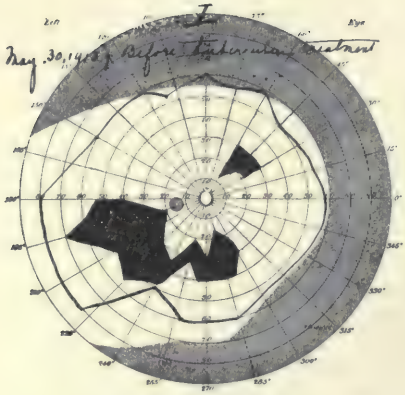
During June she had repeated injections about the cornea of one or both eyes, with vitreous clouding. Vision varied from normal to 20/40. Field was unchanged.

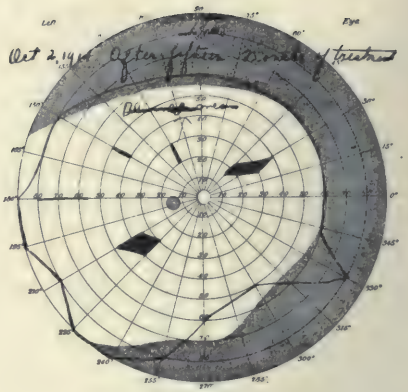
I then asked Dr. Wilson to see her with me on account of the tuberculous family history. After a thorough clinical examination, which was negative, we decided to use the von Pirquet test, recognizing, however, its small value in adults. Both of us had always been timid about producing intra-ocular reaction. This dread, at first theoretical, was confirmed by cases 1 and 3. The von Pirquet test was made by Dr. Wilson himself, and was positive. It was then decided to make no attempt to obtain focal reaction and to put Mrs. R. on therapeutic doses of tuberculin. She was accordingly given:

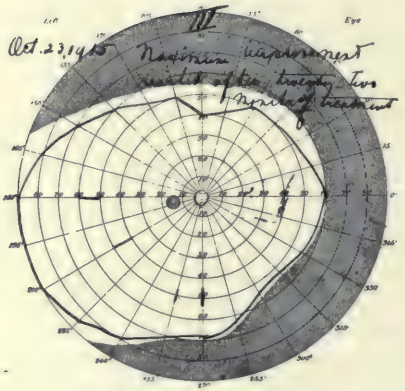
℞ Tuberculin, B. F. (human) O.001 mg.
Normal salt solution with 0.5 per cent. phenol. 10 c.c.
Label: Give 0.2 c.c. of solution once a week.

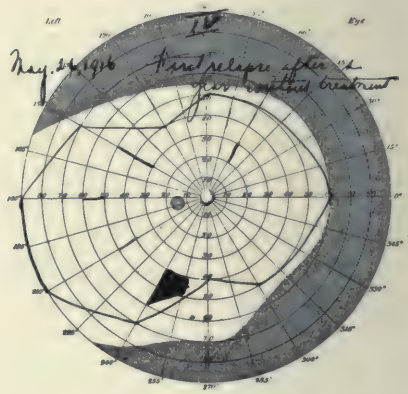
Thus, she was to get 0.00002 mg. once a week from her family physician, until further orders. This dosage was continued without febrile reaction for six months (until December, 1913), when Dr. Wilson advised addition of 0.00001 mg. to the weekly dose (0.00002 mg.) until this should reach 0.00008 mg. This was accomplished by the end of January, 1914.

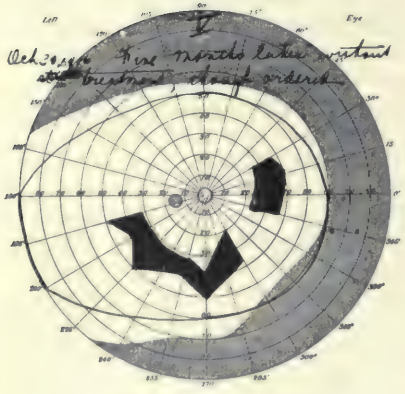
On Feb. 2, 1914, there was the first evidence of improvement, diminution in size of the scotomata. A weekly injection of 0.00008 mg. of tuberculin was continued until May 10, 1914 (ten months from the time of starting), when it was stopped because of freedom from trouble for the preceding three months. After two weeks (May 28), circumcorneal injection reappeared in the right eye. Early in June, tuberculin was renewed with the initial dose of 0.00002 mg. every ten days and increased as before. The chart, Figure 2, of Oct. 2, 1914, shows an enormous improvement in the scotomata. On December 17 this improvement had been maintained. Central vision was 20/15. Tuberculin was then discontinued. On March 13, 1915 (three months later), small scotomata reappeared. There had been occasional circumcorneal injection of either eye. Tuberculin was given as before with gradual increase to 0.00008 mg., starting April 1. She reported to me toward the last of May with a history of no further discomfort. Tuberculin was stopped. She was not seen again until

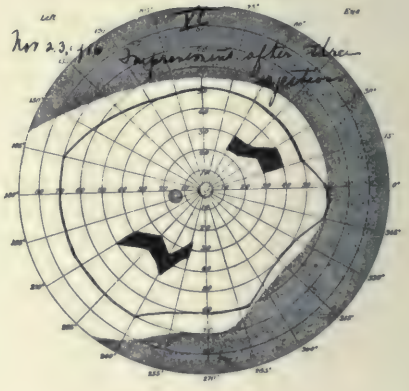

I.
Left
Eye
May. 30, 1916. Before Tuberculin treatment


Left
Eye
Oct. 2, 1916. After fifteen Doses of treatment


III.
Left
Eye
Oct. 23, 1916. Maximum improvement after two months of treatment


IV.
Left
Eye
May. 24, 1916. First relapse after 1st course of treatment


V.
Left
Eye
Oct. 30, 1916. Five months later without treatment


VI.
Left
Eye
Nov. 23, 1916. Improvement after three injections

Oct. 23, 1915, being practically five months without treatment. Her history was negative and field was normal except for blurring of the 5 mm. white disk at a few points (Fig. 3).

In February, 1916, this same condition persisted, she having been seven months without treatment, with no symptoms. However, on May 26, 1916 (about one year after discontinuance of treatment), the circumcorneal injection returned, and a small scotoma appeared in the lower and outer quadrant (Fig. 4). On May 26, tuberculin was renewed, continued for a month and stopped without orders from us. I saw her on July 21, when she urged us to go back to the tuberculin, saying she felt better when taking it. Her physician was instructed to continue it in moderate doses (0.00001 mg.) once a week during the summer. However, he went away from home and Mrs. R. received no tuberculin from July until her next visit to me, Oct. 20, 1916, when it was renewed. Her eyes were again congested and the scotomata were enlarged (Fig. 5).

She reported again on Nov. 23, 1916. She had had 0.00002 mg. of tuberculin on November 3, 11 and 18. She complained of some headache, but the scotomata were definitely smaller (Fig. 6).

Clinical examination by Dr. Wilson was negative save for a blood pressure of 200, an increase of 50 in the last three years, suggesting possibly further vascular changes. Urinalysis at the time was negative for albumin and sugar, though an examination of a twenty-four hour specimen was impossible. Dr. Wilson advised the continuation of a 0.00001 mg. of tuberculin without increase for two months, unless reaction should occur.

Points to be noted:

(1) Tuberculin treatment of eight months' duration before improvement. (2) Relapses during twenty-two months whenever injections were discontinued. (3) Then a year without symptoms and in consequence without treatment.

I have grouped these four cases because they present useful points in the diagnosis and treatment of uveitis. What is the average attitude of the ophthalmologist to the study of underlying causes in this disease? The first things he thinks of are syphilis and tuberculosis. Doubtless, there are some who still cling to rheumatism; but most of us have learned that in acute articular rheumatism we seldom see uveitis, and that what we call chronic rheumatism may be one of a great many things, the exact diagnosis often being impossible. After a Wassermann test, if negative, investigation for tuberculosis naturally follows. Here we are confronted with a practical difficulty. Ocular tuberculosis, especially of the uveal type, is usually a local lesion. Nothing positive is found by clinical examination, and we are thrown back on vaccines. Nothing short of a focal reaction is certain. Is a focal reaction safe? And how much tuberculin should be given?

As regards safety, in my first case the focal reaction promptly and permanently lessened vision and in my second case might have done the same, had it occurred nearer the fovea. The doses in these two cases were 0.5 mg. and 0.001 mg.—a wide difference. Basing my judgment on the teaching of these cases and the observations of men who are more familiar with tuberculin reaction than we can possibly be, I have come to the conclusion that the dose which will produce a focal reaction is absolutely unknown in any individual case until tried; and that, if we determine to push tuberculin until such a focal reaction is obtained, we are deliberately assuming a risk.

Again, do we need a focal reaction? In a given case, there may be a history of tuberculous exposure, local ocular lesion without other symptoms, an absence of other causes and finally a negative or positive von Pirquet. Where do we stand? It is generally conceded that two or three negative von Pirquet tests justify exclusion of diagnosis of tuberculosis. We all know that a positive reaction indicates nothing more than an infection at some time in the previous life, and may be a healed or limited lesion. But is not this all we need? Are we not safer in beginning then with minute therapeutic doses with gradual increase, until we find the patient's point of tolerance, than by insisting on a diagnostic focal reaction in the beginning? In cases three and four this course produced splendid therapeutic results.

If it be argued that we are not sure of the diagnosis and the course is tedious, I would say that, even so, it is better than damaging vision by diagnostic zeal. Of the two cases benefited by tuberculin, one has remained well while the other shows tendency to relapse when without the vaccine. It seems that tuberculin, short of curing, can supply an increased resistance; that the patient misses it, and it may be our duty to supply it by small doses for an indefinite period.

Theobold Smith[1] says: "All parasites induce in the body of the host a degree of antagonism higher than that existing before the invasion. This antagonism may not be sufficient to eliminate the parasites; it may not even be strong enough to check the march of invasion and the progressive disease resulting therefrom; it may even in some of its stages be injurious to the host. . . . In a body already infected with some other virus, living vaccines may cooperate with this virus and become pathogenic for that individual. . . . Localized bacterial infections are processes toward which the human body in general possesses a

high degree of natural resistance. . . . It is impossible to separate the toxic from the immunizing product." Finally he says: "In conclusion, let me briefly summarize the points to be emphasized. All parasites tend to increase the resistance of the host in which they live and multiply. Out of this universal fact a number of practical problems arise. In any given disease is it worth while to try to raise this immunity, and how much energy will it cost the patient? If worth while, what is the best and most sparing way of raising such immunity artificially? In any localized infection we must ask: Is this a beginning process without attendant immunity, or is it a residual process associated with general immunity? If the latter, vaccines may be considered safe. In processes associated with fever and bacteriemia, science says hands off until we know whether we have a progressive disease with gradual undermining of the resistance or a more localized affection in which the excursions into the blood are secondary. In any case, the use of vaccines in these cases must be regarded as experimental, and should not be undertaken save in conjunction with one trained in immunologic problems."

What, as ophthalmologists, can we learn from this? (1) That infection induces in the host its own antagonists, but not always enough to check the process, and that this antagonism may be in itself injurious. An obvious deduction is that we, as eye clinicians, ought to know if this latter state exists, before we induce more antagonism. What shall tell us? (2) The human body possesses "a high degree of natural resistance" to *"localized* bacterial infections." Uveitis is a localized lesion. Consequently, Dr. Smith's concluding question comes home with great force. In a given case, are we dealing with "a beginning process without immunity, or is it a residual process with general immunity?" In the former case, Nature is manufacturing antagonists. If we try to help, we may add just enough to push the patient over the damage line; for we cannot separate "toxic from the immunizing products." If it be a "residual process," that is, if there be present none of the symptoms which we call "acute," but a condition which lags, relapses and will not get well, we can infer that Nature has reached her limit. Dr. Smith shows us how the internist should go at the problem. He should watch for fever and other evidences of bacteriemia. He should give Nature all the show she wants, or she may rebel. But local eye lesions do not produce enough systemic disturbance to give this information. We are forced to reason on local conditions. To us, advance of

the process in a short time, descemetitis, intense congestion, turbid vitreous, etc., are guides. While these are advancing, the process is not "residual."

The following conclusions seem to present the proper attitude of ophthalmologists toward the use of tuberculin for diagnostic or therapeutic purposes: (1) Avoidance of focal reaction in intraocular lesions. (2) Avoidance of tuberculin as a therapeutic measure in acute cases. Under "acute" there should be included duration and advance of the lesion as indicated by condition of the vitreous, descemetitis, etc. (3) Tuberculin is indicated when the trouble is nonprogressive, but does not get well. (4) A positive von Pirquet test with elimination of other causes is sufficient basis for the use of tuberculin in therapeutic doses. (5) The present status of vaccine therapy is so uncertain in the opinion of even those most expert and experienced, that ophthalmologists owe it to their patients to conduct this treatment under the guidance of one trained in "immunologic problems."

VISUAL FIELDS IN PELLAGRA

A PRELIMINARY REPORT

F. PHINIZY CALHOUN, M.D.

ATLANTA, GA.

My reasons for presenting this subject to the Academy at this meeting are two fold: (1) To place on record my observations of certain changes in the visual field occurring in individuals having pellagra; (2) To invite the co-operation of many southern ophthalmologists who will probably attend this meeting. They will have the opportunity to study the eyes of pellagrous patients, and we wish to have our observations confirmed. I am convinced personally of the correctness of these findings, and I believe that there are definite changes in the visual field due to pellagra.

Roberts defined pellagra as "an endemic and epidemic disease, periodic and progressive in its course, and characterized by a series of symptoms involving chiefly the digestive, cutaneous, and nervous systems." Its cause is unknown, and none of the many theories which have been advanced have stood the test of time. The suggestion that unripe or spoiled maize (Indian corn), or a one sided diet (carbohydrates) produce certain chemical toxins which are responsible for the symptoms has received the most favorable consideration.

Regardless of the cause, I believe that most experts admit the existence of a toxemia, and it is this fact which largely concerns the changes in the visual field which we have been able to detect.

In making these observations, experience has taught me that only a small proportion of these cases could be examined on account of the lack of cooperation on the part of the patient. Often considerable training was necessary and much patience was required with many of them. I used the ordinary perimeter of 33 cm. radius, and Schweigger's 1 cm. test objects.[1] When a scotoma was detected it was mapped out by Peter's hand campimeter.

1. Schweigger's test objects are 1 cm. colors imbedded in the center of a black velvet square 4 by 4 cm. This black background makes the colors appear more intense, and the moving of the velvet object on the arc is noiseless, which is of great advantage in examining these neurotic individuals.

Case 1.—Mrs. J. B. M., white, aged 46, a housewife, lives in a suburb of Atlanta. She consulted me April 8, 1915, on account of defective sight of two months' duration.

There was nothing of importance in the family history. Her health had been good all of her life, except for joint pains which she termed "rheumatism." She complained of indigestion and constipation. She had been married twenty-one years, having had six pregnancies, of which the first three were miscarriages. She had not menstruated in four months. She had not complained of sore mouth and tongue, but of extreme nervousness. She did not know that the skin on the dorsal surface of the hands and forearms was rough or brown. She was not addicted to the use of drugs, whisky or tobacco. After my examination, in which I found amblyopia with scotoma, I referred the patient to Dr. S. Roberts for a general physical examination, who reported nothing abnormal except a mild atypical pellagra. The patient being well developed and presenting none of the pellagra symptoms with which I was familiar at that time (this being my first case), I admit that I was somewhat surprised at the diagnosis, but subsequent study of the disease and the associated eye changes convinced me of the correctness of the diagnosis. The urine was negative, the blood Wassermann was negative. Examination of the nose and sinuses was also negative.

Eye examination: Vision of both eyes was 20/200 unimproved. Ocular muscles were normal; pupils were round, equal and reacted to light and accommodation. The fundus showed a decided paleness of the disks in the papillo-macular bundle. Tension was normal.

Fields: A decided contraction for form and colors. In the right eye there was an absolute central scotoma for red, and a relative scotoma for green. The left field showed an absolute central scotoma for red and green, and a relative scotoma for blue. Blind spots normal.

Subsequent eye examinations have been made which show no relative change, except that the scotomas for all colors have become less absolute. The general health of the patient has improved and she has gained in weight.

Case 2.—Dr. R. W. B., white, aged 46. He was referred to me by Dr. J. C. Johnson for an eye examination. His family history was negative. Past history: He had been in perfect health until 1901, when he had typhoid fever, and since then his health had been bad. In 1910 he had grippe. In 1913 he was much "run down," but did not know cause of weakened condition. In August, 1914, developed severe attack of "biliousness," and could not eat for want of appetite. Lost twenty pounds in three months, which was regained, but general condition did not improve. In October, 1915, he lost thirty pounds, and in April an eruption appeared on his hands and wrists, at which time he first consulted his physician.

He has never had diarrhea or sore mouth; does not use tobacco and rarely takes a drink of whisky. He says he uses no drugs. A general physical and laboratory examination was made by Dr. Johnson, who reported nothing definite except that the case appeared to be a well defined case of pellagra. The nose and sinuses were negative.

Eye examination: Vision of both eyes was 20/15 with correction. The pupils, extra-ocular muscles and fundus were normal. The field showed marked contraction for form and color, with a central relative scotoma for blue and red. The blind spots were normal.

The next case was the most brilliant one of our series, for we were able to suspect the diagnosis of pellagra from the picture of the visual fields.

Case 3.—Mrs. S. C. A., aged 26, white, a housewife, residing at Heflin, Alabama, was seen June 20, 1916. The patient, who complained of abdominal pains, a vaginal secretion, backache, headache, loss of weight and general nervousness, was referred to Dr. W. S. Elkin for a pelvic operation. The examination did not reveal any abnormalities, or changes which required surgery, but as the patient complained of headache and pains in the eyes I was asked to examine her.

Eye examination: Vision of both eyes was 20/15. Extra-ocular muscles were normal; pupils were widely dilatated, but contract to strong light. Fundus was normal, except marked physiological cupping in the right eye. Tension was normal.

Field: Marked contraction for form and colors, with a relative scotoma for red and green. From this examination I questioned the patient closely and soon learned that I could not obtain an accurate history from her, but it was definitely determined that she had lost considerable weight within the last few months, and that her present nervous and mental state had alarmed the family. From a picture of the fields, pellagra was considered as a cause, and upon examining her hands and forearms I found a roughened and bronzed condition of the skin. Examinations were made by two competent internists. One unhesitatingly made the diagnosis of pellagra. The other would not so positively commit himself, without knowing the Wassermann reaction, which could not be obtained. However, he considered pellagra the most likely diagnosis.

In two other cases of pellagra, which subsequently died, I was able to demonstrate the same changes in the visual field, i. e., a contraction for form and color and a scotoma for colors, but as there were other factors present which could have produced a central scotoma for colors (tobacco, alcohol, or morphin), they are for the present omitted from this series, as the diagnosis may properly be questioned.

I could also demonstrate in three other cases of nonactive pellagra (although two were inmates of the State Sanitarium for the Insane at Milledgeville) paracentral or pericentral scotomas for colors, besides the usual contractions for form and color.

In all I have examined about forty pellagrins in various stages. The visual fields could not be obtained of those who were confined to their bed in the last stages, although a fundus examination showed a decided pallor of the disk which might properly be called a mild atrophy. There were also others whose mentality was such that no cooperation could be obtained, and it would have been futile to have attempted perimetry study. However, in every case but one (the exception being a case that had been reported "cured" for three years), when a visual field was accurately taken, I have found more or less irregular contraction for form and colors, and in several instances an interlacing or displacement of colors, principally the reds and greens.

Without presenting the histories and eye examinations, which are unimportant, I attach the visual fields of three cases which are typical and illustrate what has been mentioned.

Eye changes, such as asthenopic symptoms, circulatory changes in the retinal vessels and especially juvenile cataracts, have long been noted in pellagrins, and mention has been made of amblyopia, pale nerves and optic atrophy by various authors (Ottolenghi, Tucker, and Procopui). I am not aware that changes in the visual field have before been recorded, although Whaley of South Carolina gives as his experience that "all pellagrins are unresponsive and no field examination could be made."

What then is the explanation of these nerve changes?

Before attempting an answer, I would call your attention to the observations of Lorenz, in an article entitled "The Mental Manifestation of Pellagra," in which he compares the mental state of pellagrins with the toxic psychoses, of which alcoholism is a good example. Their resemblance, he says, is not clinical alone, but the character of degeneration found in the nerve cells of the brain is similar in both conditions. Dunlap found axonal degeneration of cortical nerve cells, which change has been similarly noted by Spiller in chronic alcoholism. Dunlap further says that "in the absence of a clinical history, we have no means of saying on pathological evidence alone whether

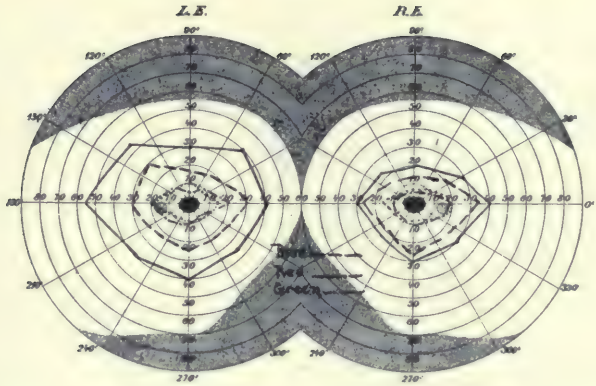


Fig. 1.—Case 1. This field shows a contraction for form and colors, with a slight interlacing or confusion of the red and greens. There is an absolute scotoma for red and a relative scotoma for green in the right eye, and an absolute central scotoma for red and green, and a relative scotoma for blue in the left eye.

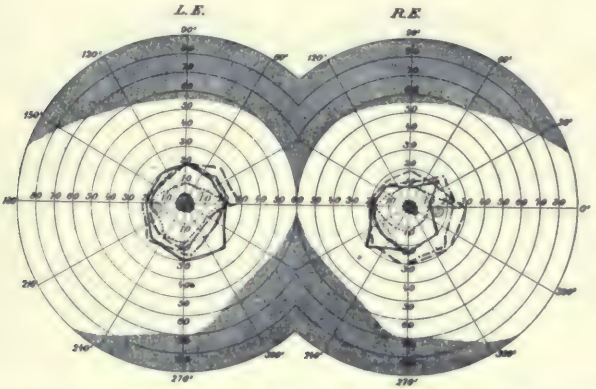


Fig. 2.—Case 2. Shows decided contraction for form with much irregularity for the colors. There is in each eye a central relative scotoma for red and blue.

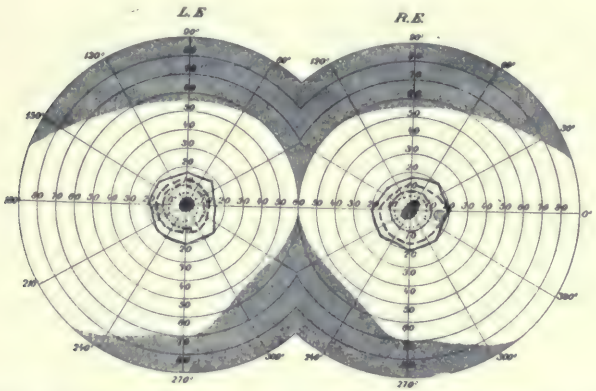


Fig. 3.—Case 3. Shows a very regular but marked contraction for form and colors, with a relative scotoma for red and green.

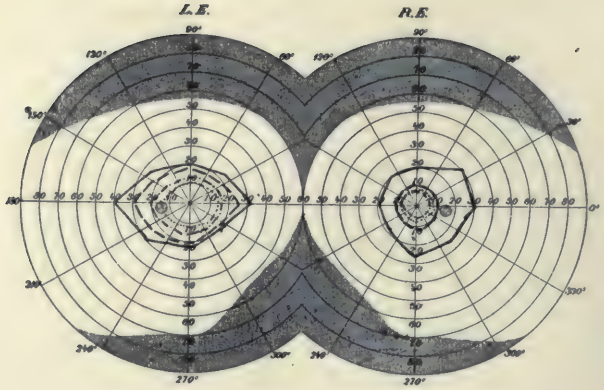


Figure 4.

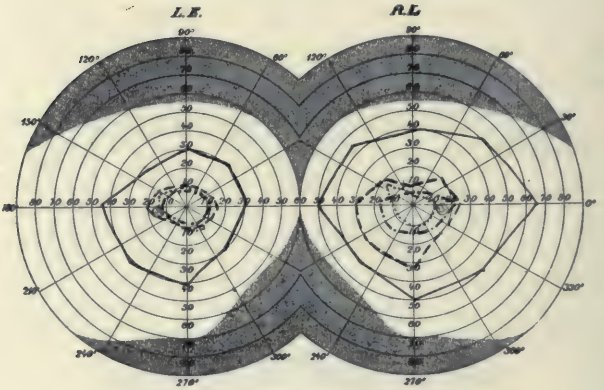


Figure 5

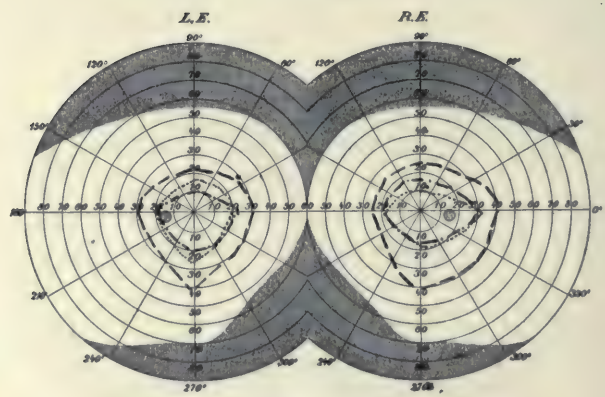


Figure 6

Figs. 4, 5 and 6.—These charts illustrate a variety of cases without changes in the central field.

a given case is one of pellagra, or of central neuritis, or of alcoholism phychosis."

Whether the direct cause for the disturbed visual field lies in this alteration of the central nervous system involving the visual tracts or centers, or in a toxic alteration in the peripheral nerves, similar to a retrobulbar neuritis produced by alcohol and tobacco, is a question which further study and investigation must solve.

In conclusion, we believe that we can positively state from our investigation:

(1) That in most cases of pellagra there is a contraction of the field of vision for form and color, with frequently an interlacing or displacement of colors, especially green and red.

(2) That in a relatively large proportion of the cases there is a scotoma for red and green and occasionally for all colors.

These scotomatous areas do not necessarily occur in the typical or acute cases, for in two patients we were able to detect these changes before a diagnosis of pellagra had been made.

BIBLIOGRAPHY

Roberts, Stewart R: "Pellagra," Mosby, St. Louis, 1912.
Ottolenghi, quoted by Marie: "Pellagra," English Trans. by Lavender & Babcock, 1910.
Tucker, R. B.: Jour. Am. Med. Assn., **16,** 249.
Procopui, Geo.: "La Pellagra," Paris, 1913.
Whaley, B. M.: "Eye Symptoms of Pellagra," Trans. Nat. Com. Pellagra, p. 229, Columbia, S. C., 1909.
Lorenz, W. F.: "Mental Manifestations of Pellagra," Pub. Health Report, 1916, **31,** No. 5, 221.
Dunlap, C. B.: "Pathological Changes of the Nervous System in Pellagra," N. Y. State Hospital Com., **7,** No. 4, 488.
Spiller, W. A.: Am. Jour. Med. Sc., Jan., 1911.

DISCUSSION

DR. H. S. GRADLE, Chicago: A study of the visual fields shown by Dr. Calhoun reveals a picture closely simulating that of a toxic amblyopia, caused by alcohol, tobacco, coffee, or other toxic medium; contraction of the visual fields in a more or less regular form; central scotoma absolute for colors, and at first relative for white, but eventually absolute for white, usually without early enlargement of the blind spot; a distinct pallor of the papillomacular bundle. This is the picture resultant from the action of a selective toxin, the site of the selection being primarily the intraneural portion of the papillomacular bundle of nerve fibers. In all probability the bundle is affected first in the vessel bearing portion of the nerve, and the subsequent degeneration advances centrifugally and centripetally at an equal rate. Immediately anterior to the entrance of the central vessels into the optic nerve the papillomacular bundle lies adjacent to these vessels in the form of a vertically oval bundle. As the nerve progresses anteriorly, the papillomacular bundle recedes from the central vessels, assuming the form of a V-shaped sector that occupies the temporal quadrant of the nerve. In this location only the tip of the V is in juxtaposition with the central vessels. Therefore, the primary toxemia probably begins at the point

of entrance of the vessels. Proceeding posteriorly from the vascular portion of the nerve, the papillomacular bundle advances toward the center and, as the canalicular portion is reached, assumes a crescentic form, lying around the central vein of Kuhnt and Vossius. This is the course as outlined by Samuelsohn.[1]

The specific toxins of pellagra are probably dialyzable and are able to penetrate the walls of their containing vessels. In consequence the optic nerve tract to be first attacked would be the papillomacular bundle or the ill-defined tracts lying on the nasal side of the vessels. In all probability, these are composed of fibers from the periphery of the retina, and any involvement of these tracts would result in a contraction of the visual fields.

This seems to me to be the most logical explanation of the eye findings in pellagra, so carefully studied by Dr. Calhoun. The idea of a central involvement of the visual tract, either toxemic or not, will not explain the clinical picture so satisfactorily as will a peripheral specific toxemia.

Dr. Luther Peter, Philadelphia: The interesting field studies in pellagra reported by the essayist illustrate well the twofold value of perimetry: first, the confirmation and elaboration of details which in a measure are visible by means of the ophthalmoscope; and, second, the diagnosis of unsuspected conditions, a careful analysis of which perimetric studies make possible.

The field changes found by Dr. Calhoun in his interesting series definitely fall into two groups; first, those caused by organic lesions in the visual path, and, second, those which are purely functional in character. In the first group are the central scotomata, contraction of form and color fields and enlargement of the blind spot. In the second are the interlacing of the color fields and the equality of the form and color fields. So far as our present knowledge goes, interlacing of color fields is either the result of inaccurate measurements or it is an hysteric manifestation. In a disease so chronic and depressing as pellagra this complication is to be expected. The second field change characteristic of hysteria is equal contraction of the form and color fields, which occurred in at least one case, that of Dr. R. W. B. The form field in this instance is smaller than the red and green, which are of about equal size. This symptom never occurs in any other condition than that of hysteria; and in all probability, if carefully sought, a typical tubular field could be elicited in this case.

The organic changes throw a definite light on the character of the lesion, at least in the visual path—a toxic amblyopia, which resembles the amblyopia of tobacco and alcohol. Most characteristic is the selection by the toxemia of the papillomacular bundle and the marked contraction of the color field. It is interesting also to note that those cases in which there was enlargement of the blind spots had also a very great shrinkage of the color fields with moderate contraction for form. From a careful analysis of these perimetric findings associated with the ophthalmic changes, it is safe to conclude that the visual changes are those of a typical toxic amblyopia, in which the ganglionic cells and their axis cylinders, which go to make up the papillomacular bundle, suffer most, although the ganglionic layer of the retina in general is involved, as shown by the marked shrinkage of the red and green fields in particular, with moderate contraction for form and blue. The hysteric manifestation is a coincidence.

---

1. Graefe's Arch. f. Ophth., 1882.

We are much indebted, indeed, to Dr. Calhoun for calling our attention to this interesting phenomenon, as it gives us not only a definite knowledge as to the ocular pathology in pellagra, but it throws a contributing light as well on some of the problems in pellagra encountered by the internist.

Dr. H. M. Lokey, Atlanta, Ga.: I am very glad to have heard Dr. Calhoun speak of this, as three years ago I made some observations of this nature, except that I did not go so carefully into the perimetric findings. The internist is still in doubt as to the cause. The poor tonsil is blamed by some—the degeneration of the products of the tonsil. Uremia was given as a cause, but certainly it is a fact that the nervous system is to a great extent involved, as manifested by the mania that comes on in many cases. I had two patients with marked contraction of the red field; neither case had been diagnosed as pellagra, but one patient died three months later with acute pellagra. The white field was normal. The other patient was a man who had never exhibited pellagra, but came in on account of his vision. The white field was normal, but the red was contracted. He had an eruption on his hand that indicated pellagra, so I sent him to a specialist. He has since died. The other cases are chronic cases. One patient whose diagnosis was pellagra had a sister who had it. She was running certain symptoms the same as her sister, but had not consulted a physician, but when I found the contraction of these fields, I advised her to go to a specialist. She was cured in a few months' time of the eruption. This is a great field of study, and after hearing this paper, I shall go more carefully into these cases myself, and I hope we can get our colleagues in the North, as well as those in the South, to do more active work along this line, for I do not believe this disease will be confined to the Southland.

Dr. F. Phinizy Calhoun, Atlanta, Ga. (closing discussion): We know so little about the subject that I am for the present willing to accept the suggestion of Dr. Gradle as to the cause of this neuritis. Since making this report I have seen a case in which the field for form and colors is not more than 10 degrees; and hysteria, as suggested by Dr. Peter, may play some part, as nervous symptoms are very prominent manifestations in the condition. We are now having several optic nerves examined, but so far we are not able to report anything definite.

CAMPIMETER VERSUS ARC PERIMETER

LUTHER C. PETER, M.D.
PHILADELPHIA

With increasing interest and an equally growing demand for careful studies of the visual field, one naturally asks how are these studies best made and what mechanical device is best adapted for an accurate and at the same time expeditious method of routine investigation? Answers to this question will undoubtedly vary greatly. Every man who has made a careful study of perimetry has found most of the instruments at his command faulty in one or more particulars and has supplemented the long list of perimeters and campimeters with variations of his own, in order to overcome what seem to him to be the greatest defects. Those who are not in the habit of doing routine perimetric work may be influenced by the equipment which they already possess and with the technic of which they are familiar. Their judgment, therefore, would naturally be preconceived. A third difficulty arises from the fact that certain definite investigations are best conducted on an arc perimeter and others are unquestionably subjects for study on the tangent perimeter or campimeter.

It is not the aim of the author to dogmatically answer these questions for the thinking man of the profession, but rather to present the reasons for his special preference.

Accuracy, practicality and simplicity of technic are the standards by which a perimeter or campimeter must be judged. Adaptability to a specific purpose is the first essential of any instrument, and the renewed interest in perimetry in recent years is due to the fact that precise and accurate information is obtained thereby. It is, therefore, of first importance that our instrument be accurate. If, however, the art of perimetry is to be practiced by all ophthalmologists, the questions of practicality and simplicity must receive due consideration. One of the greatest objections the rank and file of the profession advance to the practice of perimetry is the great amount of time required to make these studies of value. To many of our busy ophthalmologists this is a valid objection. One who would practice perimetry must necessarily be willing to give considerable of his time, for which, however, he will be amply repaid. Expedition, therefore, is an essential. Simplicity of technic may be added as a third factor, in

order to popularize so important a method of ophthalmic investigation. If the technic is easily mastered, expedition is assured.

Accuracy. — In the evolution of the modern arc perimeter, expedition and simplicity have been determining factors. In a commodious, well lighted office the perimeter may lay claim also to accuracy in outlining most normal, or nearly normal, peripheral form and color fields. Can the same be said of studies of the more important central field? I would not for a moment have the reader infer that the peripheral field studies are unimportant. In early pituitary disease, the simple noninflammatory glaucoma, and in many other conditions, the detection of slight peripheral changes is of very great value for early diagnosis. It is significant, however, that 90 per cent. of our field studies are made on well advanced pathologic cases with well defined alterations in form and color fields. The plotting of a normal form field is rarely necessary unless one wishes to do experimental work, and for such study the arc perimeter is best adapted.

After years of study and practice, however, it is the writer's conviction that the tangent perimeter will admit of accurate study of the green, red and blue fields in the normal patient and that fairly accurate information can be obtained in even very moderate contraction of a form field. If the vital question of illumination is taken into consideration the fact that the flat surface of a tangent perimeter can be properly lighted up before a window with greater ease than the hollow sphere defined by an arc perimeter, will more than counterbalance the slight inaccuracies in extremely peripheral studies on the campimeter. In measuring normal fields, illumination for white is not so important a factor; in pathologic conditions, however, it is of paramount importance. (The illumination of the test object by electricity is to be condemned, because the tests for light and color are by this method made at the same time and the results obtained are not the same as when color and light tests are made separately.)

The only claim of superiority of the perimeter over the campimeter, therefore, loses much of its value, except in the study of a normal or nearly normal form field. Studies of real value are, with few exceptions, confined within 45 degrees of the fixation point. Within this area the mechanical defects of the campimeter are a negligible quantity, and when the elasticity and flexibility of the campimeter are considered, the objection to the tangent perimeter must of necessity disappear.

The arc perimeter may be employed in determining also very much contracted form and color fields, but in this it has no advantage over the campimeter. In studies of scotomata and the blind spot of Mariotte, however, it is valueless. Its inelasticity and inflexibility here become apparent. How many of you have made satisfactory studies of the blind spot on an arc perimeter? How many of you have felt satisfied with your studies of a small scotoma in the central zone? Have you been able to definitely study qualitative color changes in this central area, and have you been able to graphically outline the areas in which such changes have been noted? I know your answer to these questions, because everyone who has done much perimetric work is acquainted with the great shortcomings of the arc perimeter.

It would be neither fair nor scientific to claim that the tangent perimeter or campimeter is without fault. The objection to the plotting of the most peripheral temporal field can never be overcome because of the inherent impossibility of approximating a flat surface to a curved surface. Within the forty-fifth degree circle of the fixation point, however, this objection is negligible.

I need not further comment on the great adaptability of the campimeter to a study of the blind spot of Mariotte, central scotomata and central color changes. The importance to the patient and physician of this central area as contrasted with peripheral changes is also equally apparent. Early cutting in the form field may be recognized on an arc perimeter, but an equally early cutting will be noted in the colors, and this color shrinkage can be outlined more satisfactorily on a tangent perimeter. In fact, if an early evidence of field change is sought, it will, with few exceptions, be found primarily in the color fields. So far as accuracy is concerned, the campimeter must be, therefore, the instrument of choice.

Practicality and Simplicity of Technic.—In order to make any method of study popular and to secure its adoption by the rank and file of the profession, such method must be rapid and easily acquired. It has always been the writer's contention that perimetric studies require much time and patience—apparently a sacrifice on the part of the busy ophthalmologist. The results obtained, however, not only justify this apparent loss of time and energy, but in many instances really demand it. It is essential that we reduce the time limit for such studies to a minimum, and this is accomplished by simplicity of technic.

Measuring the relative value of the campimeter and arc perimeter by this standard, we are again forced to a preference for a suitable campimeter. Few offices are sufficiently large and well lighted to allow the permanent placing of an arc perimeter in a well illuminated and convenient location. The result is that the instrument must be usually moved into place for each examination. Furthermore, if the examination is limited to the defining of the peripheral field the work can be done expeditiously on an arc perimeter. Our work, however, is far from being complete with these measurements alone. The silhouette method of plotting the visual field is correct so far as it goes; but it tells the observer nothing as to the process that may be in active progress in the central field, and this information has definite localizing value, whether in disease of the retina and choroid, or in the transmitting neuron in a definite part of the visual path.

The boundaries of the projected field are necessary, but an analysis of the character of the preserved field is equally important. We need a photographic view of the field that not only outlines the most peripheral boundaries, but also reveals its character — its perfections and imperfections alike. A search for scotomata and the outlining of the blind spot may be imperfectly performed on the arc perimeter, and these, at best inaccurate results, can be obtained only after a long and tiresome search, exhausting both to patient and physician. Because of this difficulty, proper examination for central field changes will be abandoned if the arc perimeter is employed and silhouette field wil be the result.

Objections to the size of the large blackboard or tangent perimeter may also be raised by physicians whose offices are not large and well lighted. The writer has solved the problem for himself by devising a "hand campimeter," which is now being generally used throughout the country. This instrument is small, light in construction, easily held in one hand by the patient, and after the technic is once mastered, admits of rapid and easy study before a window. By its aid complete studies may be made of the entire field with the exception of the most peripheral temporal and inferior form field in a normal case. Nor does it lend itself readily to a study of tubular fields. With these two exceptions, it covers rapidly and with facility the whole range of perimetric investigation, and above all with accurate results.

To the writer, therefore, the arc perimeter in its present form is an obsolete instrument, possessing no real value which may not

also be found in a campimeter, and wholly incomparable to the flexibility and elasticity of the campimeter for central field investigations. To some, my statements may sound rash and unjustifiable, but to those who have given much thought and study to perimetry and *who have actually spent much time in its practice,* they will stand as a concise summary of the relative merits of tangent and arc perimeters. The campimeter is not all that we could wish for in some branches of our study, but even in its present incomplete form it possesses so many advantages over the arc perimeter that the latter instrument may be very justly regarded as an office ornament rather than an instrument of much practical value in perimetric work.

DISCUSSION

Dr. W. B. Lancaster, Boston: Perhaps I can best open the discussion by giving my personal experience in the matter. Since I have had a Peter's campimeter in my office I have found that it tends to make the taking of fields more of an everyday matter, not reserved for infrequent cases. There are some cases in which an examination of the visual fields is indispensable, and there are border line cases in which an examination of the fields might throw some light on the diagnosis or prognosis. The easier it is to take the fields, the more of these border line cases will be examined in that way. Also the more fields we take, the more we find ourselves depending on that aid to diagnosis and the more expert we become in taking and interpreting them. Hence the first great advantage of this hand campimeter is that it is so convenient and rapid. I keep it within arm's length of my chair and can make use of it with ease. I have an adjustable table on which the patient rests the handle.

For people who are not strong it is fatiguing to hold it by the handle with no support. Better results are obtained if the patient is comfortable. You need a small test object to get the best results.

The next great advantage is that it is more acurate than the perimeter for measuring defects within 45 degrees of the fixation point. This we must admit with Dr. Peter is the area of most importance. In some cases what interests us most is the question of a slight or moderate contraction of the periphery of the field. Here we must have recourse to the perimeter.

A third advantage is the freedom of movement of the test object. The most accurate and rapid way to map out the blind spot, or a scotoma, is by moving the test object at right angles to the boundary of the spot to or from the center of the defective area. With some perimeters we are limited to movements along the meridian unless we move the whole arc, a motion which the patient too readily sees. This is an important defect of self-registering perimeters in particular.

Finally we have the testimony of the patients. It is a common experience for them to remark that they prefer this method to the perimeter, which they have had used before, by me or some one else.

Dr. Hiram Woods, Baltimore: Dr. Reber was good enough to send me a campimeter some time ago. I have found it useful in searching for paracentral scotomata. Mariotte's blind spot seems to be more easily noticed by the patient than with the arc perimeter. The patient apparently preserves central fixation with greater ease than with the arc perimeter.

We are compelled to use something to obtain excentric vision more frequently than was the case years ago. This has grown out of greater care in diagnosis. I was much impressed by a remark in Dr. Edward Jackson's book, something like this: When a patient describes his symptoms very vaguely, take his field. One is often surprised how promptly a careful study of field findings reveals the remote causes. Again, a small scotoma in the field sometimes enables us to find a fundal lesion which, in the first examination, has escaped us. It is in just this class of cases that the campimeter excels.

Dr. H. S. Gradle, Chicago: I think Dr. Peter's instrument is excellent for clinical use. It is of great value and one we should use right along. I disagree, however, as to its accuracy in showing the blind spot or central scotoma. I do not find we can make an accurate measurement on this instrument, as it is held too close. It is held 16⅔ cm. At that distance in hyperopia and presbyopia we get a marked blurring. I use a different type and at 60 cm. Mine is in the form of a solenoid covered by a sheet of celluloid. Two arms against the posterior surface of the celluloid sheet hold ball bearings against the anterior surface. One is the fixation point and the other the test object. It is ruled off into centimeter sections, the horizontal ones lettered and the vertical ones numbered. We can transcribe that in any form we wish for preservation. By using greater distance and having nothing visible moving but the test object, and introducing a secondary fixation mark, we are able to do much more acurate work as regards the blind spot and central scotoma. For rough work this is not essential.

Dr. Wendell Reber, Philadelphia:: The facts set forth by Dr. Gradle are true, but what we are looking for is an instrument we can use every day quickly in our everyday practice. Gradle's instrument is the most admirable thing for fine work, and in the sense that it places nothing but the test object in the field of vision, it is a splendid idea. It is true we use this at short range. In assuming the blind spot it will not matter. The question is whether it would not be too small. If you use a 2 mm. object the color sense begins to fall off, and I believe the accuracy is just so much less. If I cannot bring the vision up to normal, I reach for the hand campimeter and begin to assume what size the Mariotte spot of the presbyope would be. The objection to the majority would be that we furnish something too large for such examinations as we wish to make. A 5 mm. or 2 mm. object is best. A further objection to the arc perimeter is that the carrier itself is seen moving along before the object comes into view and the patient can hear it coming. I think the next improvement will be an umbrella.

Dr. Patton, Omaha: I have nothing to add beyond my enthusiastic endoresement of Dr. Peter's campimeter. After a year I find it most practical and valuable. It has increased our perimetric work ten fold, and is a most valuable addition to our armamentarium.

THE EXCISION OF THE TARSUS IN TRACHOMA*

GEORGE FRANCIS SUKER, M.D.
CHICAGO

In this country the excision of the tarsus, together with the overlying conjunctiva to within 1 or 1.5 mm. of the margin of the lid, and the covering of this area by conjunctiva drawn from the upper retrotarsal culdesac is not practiced as much as abroad.

For this there are several reasons: principal among which is the dread that an entropion may ensue; another, when the tarsal space is not covered by conjunctiva, it is filled in with granulation tissue which in time contracts, eventually producing an entropion; finally, some fear that after an excision of the tarsus the regular contour of the lid edge may become more or less distorted. Then, too, that a veritable lagophthalmus may result from the excision of the tarsus, because of the undue retraction, has deterred some from availing themselves of this useful and beneficial operation. That also a possible ptosis may result from undue damage to the levator has been a drawback to the operation as ordinarily performed.

No doubt, unless a strict surgical technic is observed and the proper judgment exercised in determining how far one should remain away from the free border of the lid, and how much conjunctiva is to be sacrificed, and how far to carry the dissection up into the retrotarsal fold, some one or more of these supposed untoward conditions may possibly arise.

Another factor which deters some operators from employing this operation is their indecision in the proper selection of cases. This latter difficulty is not a hard one to surmount.

On general principles, it may be stated that the operation is indicated in:

(*a*) Any case of trachoma which does not yield to the usual line of treatment, no matter how vigorously or conscientiously the same has been executed;

(*b*) In cases of so-called "hot box"—i. e., in cases wherein the tarsus is extensively cicatrized, uneven, and more or less studded with islands of vascularized pinhead nodes, which

* From the ophthalmic service in Cook County Hospital.

constantly irritate the cornea and cause a persistent but attentuated pannus. The tarsus itself may not be greatly incurvated;

(*c*) In cases in which the tarsus is comparatively smooth, yet greatly incurvated through cicatrization, thus exerting undue pressure upon the cornea, causing ulcers in the same and vascularization at the limbus;

(*d*) In cases of a velvety infiltrated tarsal conjunctiva with more or less extensive pannus, such as are often seen in the old exuberant chronic trachoma, which apparently yielded nicely in the acute stages, but in which the too heroic measures have set up this proliferative and velvety conjunctiva which ultimately cicatrizes extensively and incurvates the tarsus;

(*e*) In such cases in which the upper half of the cornea is frequently subjected to ulcers after an apparent diseappearance of the trachoma;

(*f*) In such cases in which the apparent cure resulted in producing an entropion with its usual train of symptoms;

(*g*) In such cases in which the pannus does not satisfactorily subside, though the tarsus and retrotarsal folds are seemingly in a fair state of aggravation, as it were, to either cause or maintain said pannus;

(*h*) In such cases in which the tarsus was grattaged and left distorted and studded with connective tissue pinpoint nodules.

On the other hand, it may be stated, in broad terms, that the excision of the tarsus is not indicated when the cornea is not extensively involved, either as an acute affair or a chronic one.

The presence of corneal ulcers is no contra-indication for delaying excision of the tartus; on the contrary, it is an indication for the immediate performance of it.

As long as the cornea remains free from so-called gravity ulcerations at the pupillary margin of the pannus and this pannus is apparently quiescent, the tarsus need not be excised. Should, however, this pannus give the appearance of a marginal infiltration or thickening at its pupillary border, then the tarsus should be excised. In these cases the tarsus may be, and often is, in a state of essential cicatrization with minute connective tissue ridges or rugæ and yet appears relatively smooth, still causing sufficient friction to the cornea.

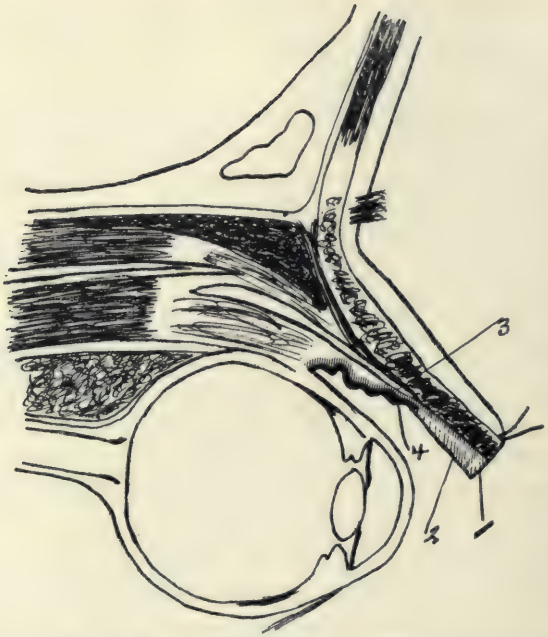


Fig. 1.—1. 1½ mm. of tarsus left at the ciliary border. The cut is made perpendicular and parallel to lid edge. 2. Portion of tarsus to be excised. 3. Line of incision and its direction from ciliary edge to culdesac. 4. Retrotarsal conjunctival fold.

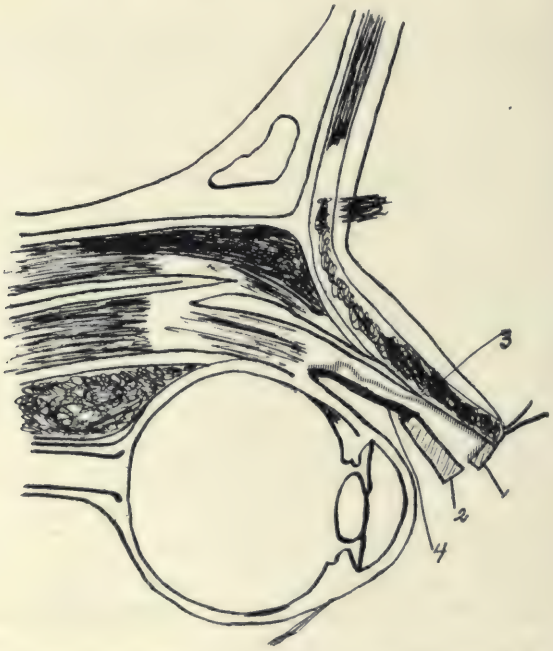


Fig. 2.—1, 2, 3, 4. Same as in Figure 1. After the incision is made.

Given a case suitable for excision, in order to obtain the best surgical and therapeutic results, the following suggestions are to be observed:

(1) Always leave a 1 to 1.5 mm. strip of the tarsus at the ciliary margin of the lid. This strip of tarsus is seldom if ever involved in the trachomatous process (Figure 2).

(2) Begin the excision at the ciliary border of the lid—not at the retrotarsal border (Figures 1 and 2).

(3) Do not ever attempt to save the conjunctiva directly overlying the tarsus.

(4) Do not excise more than just the tarsus.

(5) Dissect the conjunctiva far enough, throughout the entire width of the lid into the retrotarsal fold, taking care not to have much more than just sufficient conjunctiva to comfortably cover, without much tension, the area formerly occupied by the tarsus (Figure 3).

(6) Do not excise tarsus until the retrotarsal fold has been dissected as mentioned. This avoids undue manipulation with forceps of the edge of the conjunctiva to be attached to the ciliary edge of the tarsus (not at all an unimportant step in the operation).

(7) Secure a good approximation of this edge of the retrotarsal conjunctiva to the 1 or 1.5 mm. tarsal strip left at the ciliary margin of the lid.

(8) Employ a mattress suture for securing this requisite coaptation just mentioned (subconjunctival and subtarsal, Figure 5).

(9) Obliterate the "dead space" necessarily produced between the lid and the fold by the stretching of this retrotarsal conjunctiva, by two properly placed sutures at a point corresponding to the previous retrotarsal border of the tarsus (Figure 4). This will transform the retrotarsal conjunctiva and lid into one, and with sutures as mentioned at the former retrotarsal edge the lid will not retract nor will entropion take place, as the anatomical insertion of the levator is thus properly maintained and ptosis does not supervene. Traction upward is exerted from this newly established line of cicatrization.

(10) Do not attempt to do this operation under a local anesthetic; under local anesthesia you cannot get the relaxation of tissue as under a general anesthesia. Then, too, the infiltration caused by the local anesthetic injection is very annoying.

Fig. 3.—1. Strip of tarsus at ciliary border. 2. So-called dead space between skin (lid) and retrotarsal conjunctiva. 3. Conjunctiva brought to lid margin (strip of tarsus). 4. Retrotarsal conjunctiva brought down after dissection as far as possible.

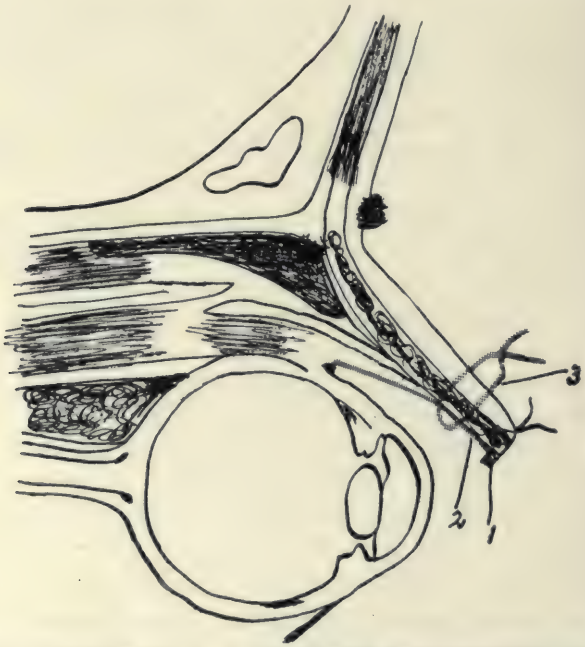


Fig. 4.—1, 2, same as Fig. 3. 3. Suture passes from conjunctival surface of lid to skin surface and tied over small pledgets of cotton. Two of these usually necessary (placed at inner and outer third of lid).

After the operation, apply a goodly quantity of sterile vaselin or White's ointment and then apply a rather snug bandage. As soon as the patient recovers sufficiently from the anesthetic, apply an ice bag directly over the bandage, which must not be too voluminous. Keep this ice bag on for several alternating hours for at least twenty-four or forty-eight hours. By doing this you considerably lessen the surgical reaction.

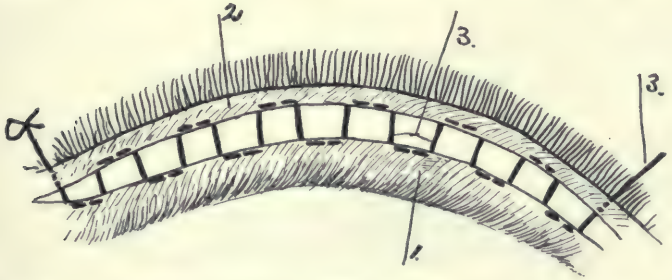


Fig. 5.—1. Conjunctiva (tarsus excised). Tarsal strip, cut surface. Course of suture. Pass the suture from the skin surface, at either inner or outer canthus (where it is fixed or tied on the skin surface), through the tarsal strip and then into the substance of the conjunctiva, back into the substance of the tarsal strip (from the cut edge) and back again into the conjunctiva. Continue in this manner until the whole margin of the conjunctival flap is neatly approximated to the cut edge of the tarsal strip. The last stitch passes out through on to the skin—tension is made so as to take up any slack. It is now suitably anchored at this point.

The sutures which are needed to establish the new insertion for the levator and obliterate the dead space (see Figure 4) can be removed on the second or third day after operation.

The mattress suture (Figure 5) along the lid edge can be safely removed on the third or fourth, but not later than on the fourth day subsequent to the operation.

Fig. 6.—Tarsal clamp. Within the fenestrum the tarsus is dissected from the ciliary border backward towards the retrotarsal fold. Before releasing this clamp, the T forceps are applied to the dissected tarsus and then the tarsal clamp is slipped over the T forceps for the continuation of the dissection.

The lid clamp and forceps which have been found useful in this operation are simple and readily applied (Illustrations 6 and 7).

The accompanying illustrations perhaps convey a better idea of the technic than a verbal description.

It is advisable, after the removal of all sutures, to use moderate astringents on the upper as well as the lower conjunctiva. For, even after the removal of the tarsus, the retrotarsal fold is usually more or less subacutely inflamed and needs attention. This is a point often lost sight of by those who think the removal of the tarsus is a panacea. Within a very short time, under the judicious employment of astringents, massage, and the like, the conjunctiva becomes very smooth and remains so.

It is not the lower conjunctiva in trachoma that is the *bête noire*, but the upper. And, as a rule, the trachomatous process is seldom as pernicious as in the upper lid.

Fig. 7.—Two of these T forceps are very convenient for the excising of the tarsus. One is applied to the portion of the tarsus after the same is dissected down from the ciliary border to the retrotarsal edge in the fenestrum of the tarsal forceps—when the tarsal forceps are unlocked and slipped over the T forceps. The second T forceps is now applied to the lid. Then an assistant can hold both and the operator dissect farther into retrotarsal fold without the use of other forceps. Thus the T forceps answer a triple purpose—control hemorrhage, avoid undue manipulations with other forceps, and make the dissection easy in so far that the whole width of lid and tarsus is within their grasp. When the dissection is carried sufficiently far, then the tarsus is excised and both forceps released.

In my service at the Cook County Hospital, my assistants, Drs. Coppes and Carr, have performed many such an excision with practically universal satisfactory cures—i. e., brought the process to an end and aided materially in minimizing the disastrous corneal opacities and pannus and doing away with the ever recurring faceted ulcers in the cornea. In each instance, the annoying little "comebacks," as they might be called, were done away with. The expression "annoying little comebacks" in old pseudo-active trachoma is more or less apropos and is readily understood by all.

In conclusion, it may be stated that perhaps there are several new steps in performing this operation which differ from the methods usually described. And since we have adopted these modifications in my service, the excision of the tarsus for conditions as above detailed has proven the only satisfactory operation.

DISCUSSION

Dr. W. B. Lancaster, Boston: I was not intending to discuss the paper, as it is not an operation I have done, but I am a good deal interested in it. I would ask Dr. Suker why he objects to leaving some of the conjunctiva?

Dr. R. H. T. Mann, Texarkana, Texas: I should say that this is a most useful operation and it seems to me that it has settled the question of trachoma. Dr. Suker did not mention it in his paper, but I do not believe the operation should be done during an attack of acute inflammation, but that it should be delayed until this has subsided somewhat. The patients on whom I have operated during the acute inflammation have been greatly delayed in recovery. Those postponed until the acute attack had subsided have recovered rapidly. I believe the operation can be done with cocain, as I have done many times, and with no deformity.

Dr. Patton, Omaha: I am much interested in Dr. Suker's paper. I should like to ask what suture material he uses, and whether this operation is applicable to cases in which there is a marked atrophy of the retrotarsal folds?

Dr. John Green, Jr., St. Louis: It seems to me this operation greatly helps in bad cases in which less radical measures are of little value. I have resorted to tarsal excision in very old cases with recurrent ulceration of the cornea. The technic I have followed is that suggested by Claibourne of New York, in which a clamp forceps of ingenious design enables one to make the incision accurately by following the curve of the horseshoe shaped blade. All patients operated on whose later history I have been able to follow have done exceptionally well. I recall in particular a woman who has been under my care off and on for ten years. Her lids were rolled twice and a total peridectomy was performed for recurrent pannus. However, under continuous treatment ulcers would now and then occur. Ten months ago I performed tarsal excision, and she has not had a corneal ulcer since.

Dr. Suker (closing discussion: In answer to Dr. Lancaster's question, I will say that formerly I tried to save as much tarsal conjunctiva as possible, but invariably failed to make a satisfactory dissection so that latterly I have given it up altogether and now sacrifice the entire overlying tarsal conjunctiva.

It is quite essential that the retrotarsal fold of the conjunctiva after the excision of the tarsus be manipulated as little as possible with forceps.

For suture material, I have been using the white twisted Japanese silk. It can be readily sterilized and the individual strands are of remarkable tensile strength, when but a very, very fine thread is needed. It can easily be untwisted—easily threaded into the needle and does not slip upon tying.

In answer to Dr. Patton, when the retrotarsal fold has been practically destroyed by the trachomatous process, I still excise the tarsus, provided I have on hand at the hospital a case for circumcision. This mucous membrane makes a good substitute.

The presence of corneal ulcers is no contra-indication, but much rather an absolute indication for a tarsus excision.

IRIDOTASIS IN GLAUCOMA

EDWARD STIEREN, M.D.
PITTSBURGH

Iridotasis, literally, stretching the iris, is an operation devised by Dr. Johan Borthen[1] of Bergen, Norway, and is an improvement on Holth's operation, iridencleisis, which was designed to produce a fistulous opening between the anterior chamber and the subconjunctival lymph-channels by incarcerating a flap of the iris in a cornea-scleral incision and allowing the conjunctival flap to heal over it.

Borthen had operated on twenty-six patients with glaucoma after the Holth method. He says that he had always doubted the advisability of incising the iris and incarcerating a tongue of iris tissue. By an inclusion of a fold of the iris, so arranged that its posterior surface coalesces with the subconjunctival tissue, a position of the sphincter external to the corneal-scleral section is assured, and with this, free drainage.

This modification of Borthen has the distinct advantage over Holth's original operation that it eliminates a difficult step of the operation, incision of the iris, which requires skilful technic and a certain amount of additional time.

Borthen compared his series of twenty-six cases operated on after Holth's method with a series of fifty cases operated on after his own method. He is convinced that his simplified procedure is better and that the incision of the iris, which was supposed to be of so much importance in the formation of a permanent fistula, has, on the contrary, the effect of diminishing the prospects of permanently reducing tension.

Holth described his operation in May, 1907; Borthen in July, 1911.

David Harrower of Worcester, Mass., was among the first to take it up in America, having operated on two patients after Borthen's method in the summer of 1912, making them the subject of a paper which he read before the American Ophthalmological Society in May, 1913.[1] Two years later he reported seven additional cases before the same society, stating that the

1. Archives of Ophthalmology, Vol. XL, No. 4.

results were invariably so satisfactory that he had since then done no other operation but iridotasis for chronic glaucoma.

Dunbar Roy[2] reported nine successful operations and strongly recommends iridotasis where no contraindications exist, for both acute and chronic glaucoma.

Verhoeff, in discussing Harrower's second paper said: "I shall continue to use this procedure as a substitute for the trephine operation until I find some objection to it of which I am not at present aware."

Harrower, in closing the discussion, said that he had received a communication from Dr. Borthen, in which the latter said that he had done 206 operations after his method, since the last report, and that every one had been successful.

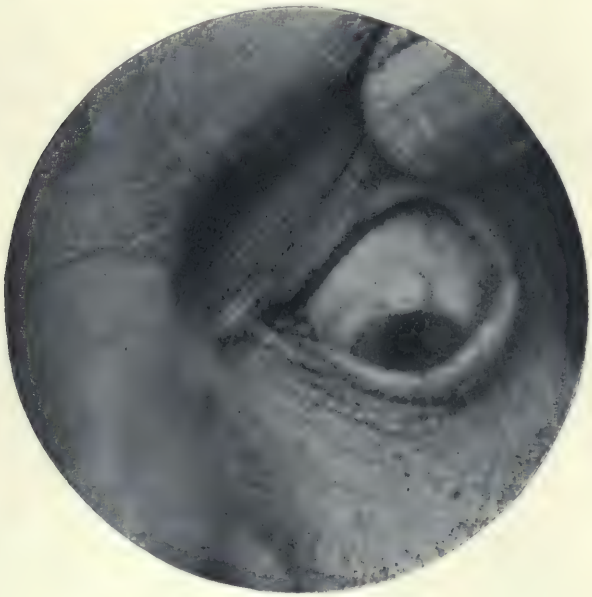


Fig. 1.—Iridotasis (Harrower).

There appears to be only one contraindication to the operation, and that is when the iris is so atrophic or friable that it cannot be drawn into the wound, as it tears into shreds with each grasp of the iris forceps. I met such a condition once in the iris of a young myope with glaucoma.[3] Verhoeff[4] states that a similar condition of the iris obtained in a patient with chronic glaucoma on

2. Ophth. Rec., 1916, March.
3. Stieren: Glaucoma with Atrophy of the Iris, Penna. Med. Jour., Vol. 18.
4. Loc. cit.

whom a simple extraction had been performed years before and on whom he attempted iridotasis.

The operation is very simple: The eye is cocainized thoroughly and a conjunctival flap made as in the trephining operation. The flap need not be so large, nor is it necessary to carry the dissection to the corneal layers, but the flap should contain all the subconjuctival tissue down to the sclera. A small keratome incision is then made under the conjunctival flap, just behind the limbus, and just large enough to admit the iris forceps. The pupillary edge of the iris is grasped and gently withdrawn, and, when released outside of the corneal incision, it will be noticed that the under-surface of the iris is now up. The conjunctival flap is stroked back into place and the speculum carefully removed, the operation requiring not more than five or six minutes to perform. Borthen recommends that a drop of 1 per cent. atropin be instilled into the eye a half hour before the operation, so as to prevent the iris falling back into the anterior chamber. This I feel is unnecessary if the corneal incision is small, not over 5 mm. in length.

The recovery from the operation is prompt. In twenty-four hours the conjunctival flap is attached and there is no need to keep the patient longer in bed. A distinct bleb forms which can be reduced by pressure with a flat instrument or through the lids. The prompt relief from pain in the chronic blind eyes is almost miraculous. In the less advanced cases an immediate enlargement of the visual field is noted, and usually central vision is likewise improved.

The prompt results following iridotasis are due as much to the stretching of the iris, thereby keeping open the filtration angle at this site, as to the subconjunctival drainage.

In this respect iridotasis differs from all other operations devised for the relief of increased intra-ocular tension.

I have performed the operation six times in four patients.

Case 1.—J. D. W., retired glass manufacturer, aged 66 when glaucoma was first noted, Feb. 23, 1915. R. E. 6/8 —, L. E. 6/16, each with + 2 D.Sph. Shiötz tonometer, R. E. 50 mm., L. E. 57 mm. The right field was moderately contracted; the left field was markedly contracted, especially below. Both disks were cupped, excavation of left extending to temporal edge of disk. Physical examination was made by Dr. T. W. Grayson. Patient's height is 5 ft., 6 in.; weight, 208 pounds. Blood pressure was: systolic 165, diastolic 110. Urine was negative. Bowels were sluggish. Eserin was ordered. Habits and diet were regulated.

March 15, 1915, R. E. 6/6 —; L. E. 6/25. Right tension was 40 mm. Left tension was 46 mm.

June 22, 1915, the vision had remained the same in each eye, but fields had narrowed. Right tension was 35 mm. Left tension was 47 mm. Eserin and pilocarpin administration with massage of eyeballs have been faithfully carried out.

July 13, 1915, iridotasis was performed on left eye. The field immediately improved as illustrated in the chart. The vision gradually improved to 6/16 and has remained so.

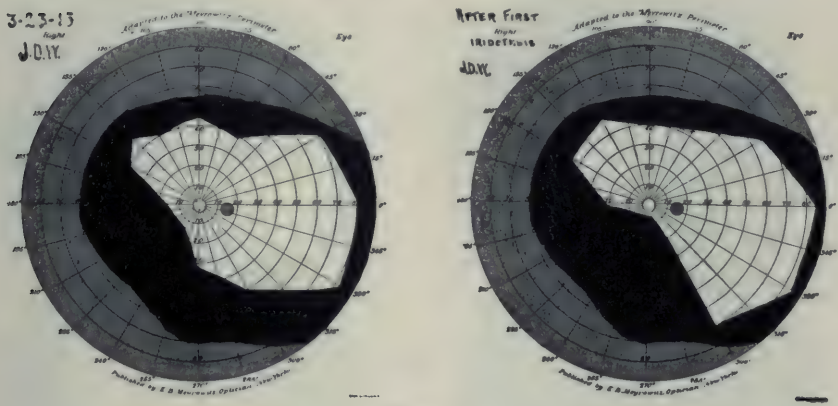


Figure 2.

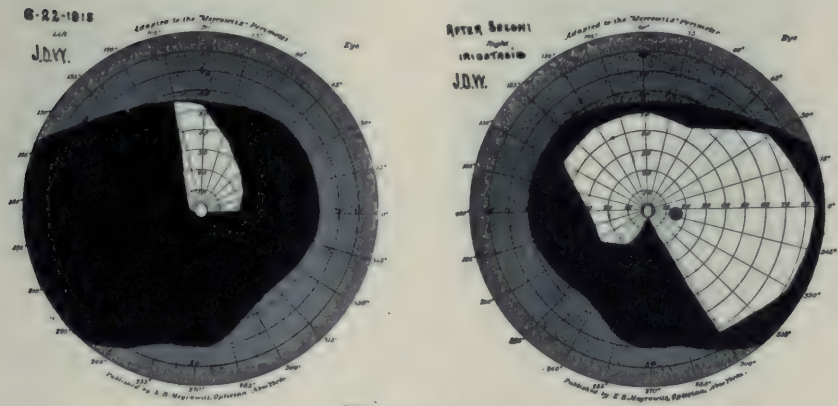


Figure 3.

Aug. 10, 1915, the right tension was 38 mm., the left tension was 24 mm. Within the next two months vision in the right eye had declined to 6/36 — and the field had diminished steadily in size.

Oct. 21, 1915, iridotasis was performed on the right eye, resulting in immediate enlargement of the field and increase in vision to 6/25. Tension was 24 mm.

In speaking to Dr. Harrower of this case six months later in regard to the slight improvement in vision in the left eye he suggested that an iridotasis downward could do no harm, which accordingly was done May 26, 1916, but there has been no further improvement in vision, although the field has enlarged following the second operation.

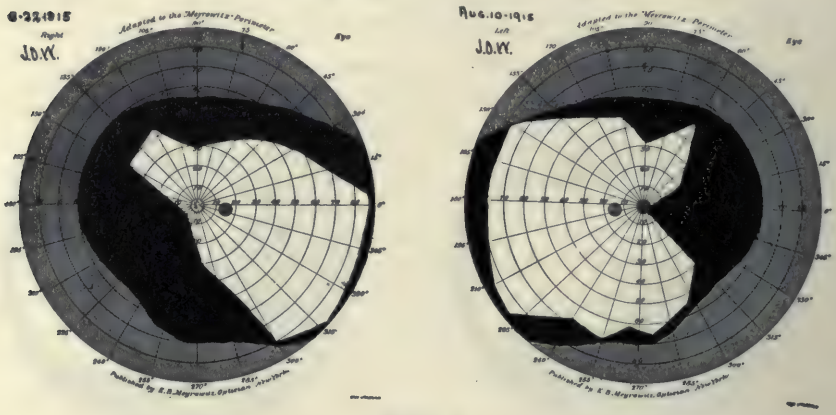


Figure 4.

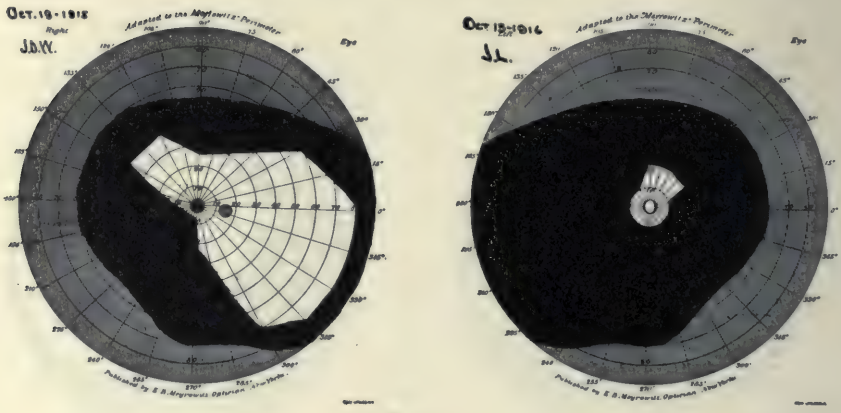


Figure 5.

The cornea, lens and media are beautifully clear in both eyes, but there is an almost total excavation of both disks.

Vision in both eyes fluctuates between 6/20 and 6/16, and the fields are good. The patient plays a better than average game of golf. Near vision is obviously limited.

Case 2.—Dr. C. B. K., a retired physician, aged 76, came Aug. 18, 1915, for treatment, when acute glaucoma developed in the left eye. Pupil was widely dilatated. There was well marked

circumcorneal injection and steamy cornea. The tension was 60 mm.

Physical examination was made by Dr. J. P. McKelvy. His height was 5 ft., 8 in., and his weight 180 pounds. The blood pressure was: systolic 210, diastolic 95. There was well marked arteriosclerosis and slight albuminuria with hyaline casts.

Patient was put to bed, diet regulated and therapeutic and hydrotherapeutic measures adopted to reduce the blood pressure. Eserin and massage of eyeball failed to relieve the pain in the eyeball and temple.

Iridotasis was performed under cocain Aug. 23, 1915. The patient experienced slight pain when the iris was grasped in the forceps. The operation was followed by complete relief from pain, and there has been no recurrence since.

November 1, 1916, the tension was 28 mm. The eye remains perfectly comfortable.

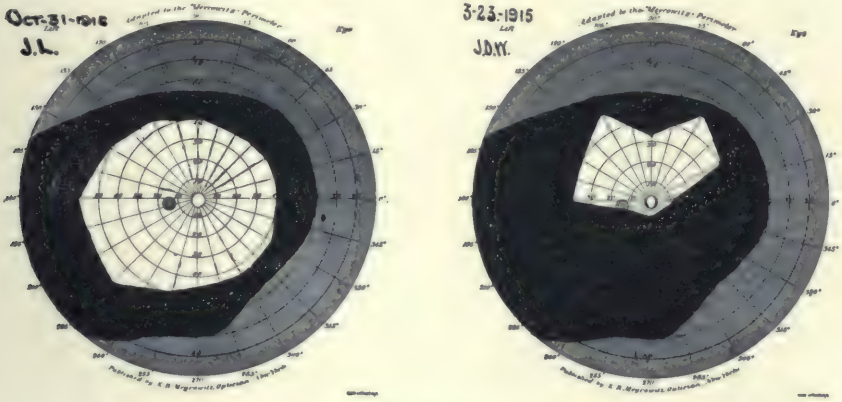


Figure 6.

Case 3.—Through the courtesy of my associate, Dr. Van Kirk, I saw D. J. C., aged 70, Oct. 23, 1916. He had had poor vision in the left eye for about two years. During the past summer he had had pain in the eye at various times and vision had gradually failed to light perception.

When seen in consultation with Dr. Van Kirk, the left pupil was largely dilatated and the cornea was faintly hazy. There was marked circumcorneal injection. The eye was stony hard, and there was no light perception. The pain in the eyeball was severe and radiated to the temple. The blood pressure was: S. 310, D. 195. The weight of patient was about 130 pounds. There was marked arteriosclerosis.

Eserin and dionin for twenty-four hours giving no relief, iridotasis was performed under cocain. Some pain was experienced when the iris was grapsed with the forceps, but the operation was successfully terminated. The glaucomatous pain was immediately relieved and has not recurred since.

November 29, 1916, the tension was 26 mm. Light perception was noted, the eye was comfortable.

Case 4.—From the Eye Clinic of the South Side Hospital. J. L., aged 70, was first seen Oct. 13, 1916. His height was five feet; his weight 118 pounds. The blood pressure was: systolic 180, diastolic 125. The left pupil was widely dilatated, cornea being faintly clouded. There was slight pericorneal injection. The tension was 50 mm. The vision was 6/40 —. The field very much constricted, as shown in chart.

Eserin was ordered, to which the iris promptly responded and the corneal haze cleared. There was no improvement, however, in vision or field. The eye was painful at times.

Iridotasis was performed Oct. 24, 1916, and was followed by enlargement of field in all meridians, as illustrated in the chart, and by improvement in vision to 6/16 +.

DISCUSSION

Dr. Adolph Krebs, Pittsburgh: I do not feel competent to say much about the sucess or advisability of this operation. I have performed it as described by Dr. Stieren on three patients with simple noninflammatory glaucoma, two aged 55 and one 78. All obtained excellent results, especially as to the reduction in tension, as in none of them has it ever reached even 20 mm. since the operation. Case 1. A male patient, aged 55, had the disease only in one eye. Preservation of the contracted field and fair vision resulted. Case 2. A female patient, aged 55, had both eyes affected, the fields being contracted to about 15 degrees from the fixation point. I had performed an Elliot trephining on the left eye with good results, however, with rather severe reaction; but the iridotasis performed several months later on the right eye was as efficient in reducing the tension, while the postoperative reaction was much less. No increase in the size of the fields has taken place, but good vision has been preserved. Case 3. A male patient, aged 78, had bilateral glaucoma, which was discovered accidentally, there never having been any symptoms of the disease. The field of the right eye was contracted to 10 degrees from the fixation point, but still had normal central vision. The average tension had been 48 mm., the disk pale and deeply excavated. Iridotasis was performed two years previous. There has been no further contraction of the field, the tension remains normal, but the vision has slightly deteriorated, due probably to senile change at the macula. The left eye has not been operated on, as it has been possible to keep it from failing by the use of miotics. I have been impressed by the prompt recovery from this operation and the entire freedom from subsequent irritation. I believe that postoperative infection is less apt to occur from this operation than after trephining.

REPORT OF A CASE OF PARINAUD'S CONJUNCTIVITIS

HOWARD V. DUTROW, M.D.,
DAYTON, OHIO

I wish to offer an apology to the members of the Academy who have seen a number of this class of cases, and who are therefore thoroughly familiar with this disease. It is not a common disease and for that reason I felt that this case should not go unreported. I have personally never seen a photograph or illustration of the disease in any textbook, monograph, or reprint, and I am sure there are a number of men present who have never seen a case. This case doubtless would have been treated by me improperly diagnosed had it not been for my good fortune to be a guest of Dr. Peter A. Callan at a meeting of the New York Ophthalmological Society in January, 1911, when Dr. R. G. Reese reported a similar case that had been referred to him from Virginia.

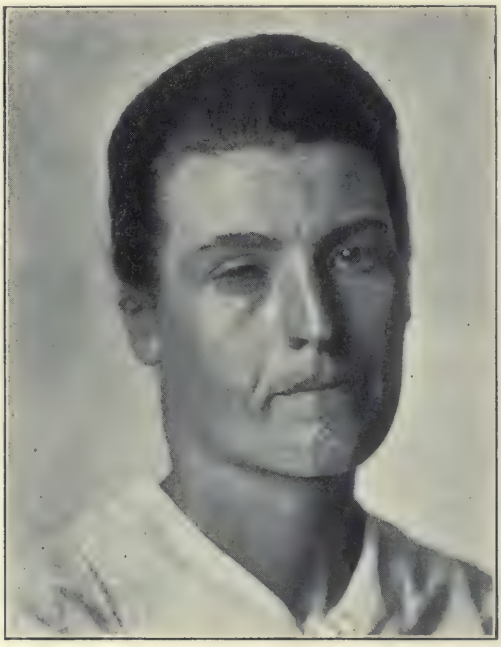
CASE 1.—Mrs. A. W. J., Route No. 3, West Alexandria, Ohio, aged 28, white, was referred to me by Dr. J. B. Lucas on June 28, 1916, with what he pronounced a purulent conjunctivitis of the right eye. Her family history was negative. About seven weeks before, her right eye had become red with a rather copious muco-purulent discharge. About three weeks after the onset of the conjunctivitis, it was noticed that the glands on the right side of the neck were swollen and quite painful on pressure. During these seven weeks her family physician ordered the eye flushed two or three times daily with a saturated solution of boric acid.

Examination showed slight puffiness of the eyelids. The conjunctival sac contained a small amount of purulent secretion. There was an excessive amount of lacrimal secretion without photophobia, a slight chemosis of the bulbar conjunctiva with rather marked injection with granular formations on the upper palpebral conjunctiva. These granules were irregular in size, some being quite large with smaller and more numerous ones interspersed. All the granules were of a decidedly reddish color, more or less transparent on oblique illumination. There were no erosions or ulcers present.

The left eye was entirely normal. There was a marked pre-auricular and cervical glandular involvement, as the photograph well illustrates. The patient stated that these swellings had been rather painful at first, but at this time were not especially sensitive on palpation. There were no areas of necrosis, but rather a

firm indurated adenomatous condition. The adenitis, I believe, is pathognomonic of Parinaud's conjunctivitis. It is the only conjunctival infection in which we have a secondary cervical adenitis of the corresponding side.

The bacteriologic examination made by Dr. M. B. Floyd was as follows: "Cultures and smears were made from the conjunctiva. The smears appeared sterile, a few polymorphonuclear leukocytes were found containing no organisms. Cultures were made on blood serum, starch agar, and plain agar. No growth was noticed until after forty-eight hours in the



incubator at 37 degrees C. all media presented a few colonies, bearing a close resemblance to each other. The colonies were elevated, clean cut, and sharp edged, the center being a little darker and thicker than the periphery. Organism stained rather slowly with alkaline methylene blue and was gram-positive. Stained specimen showed many short rods arranged singly or in short threads. Organisms unstained showed marked Brownian motion, but no motility. Smear and culture show no gonococci, bacillus xerosi, pneumococci, bacillus pyocyaneous streptococci, bacillus of Weeks, Morax-Axenfeld, staphylococci, micrococcus catarrhalis or bacillus diphtheria."

The short rod-shaped organisms to which Dr. Floyd referred may be the causative organism in this disease. The laboratory test was not concluded by the isolation and growth of the organisms and animal experimentation.

Rolandi is of the opinion that it is not a distinct morbid entity, but represents rather a clinical syndrome and in its classic form is a special type of tuberculosis of the conjunctiva characterized by an exceptional benign course and by spontaneous recovery. In conclusion, he states, however, that it has not yet been sufficiently demonstrated whether this tuberculous form is due to human or bovine tuberculosis.

The fact that this case and practically all of those previously reported have occurred among people in the rural districts who work around domestic animals, especially cattle, is sufficient evidence that they are a possible source of infection. This patient lived on a farm where there were horses, cattle, hogs, and dogs, but no sheep or cats. It was her daily custom to milk several cows twice a day, which brought her into very close contact with this source of infection.

The distribution of cases seems to be uniform throughout the temperate zone, and, as previously stated, confined almost entirely to the rural districts. I feel that there is no doubt as to its clinical entity and that it is due to a specific organism which so far has not been recognized.

The prognosis is good, though during the duration of the disease may be very much prolonged, extending over a period of six months or more. The duration in this case was about three months, without suppuration of the glands and without any permanent injury to the eye.

The treatment was very simple, consisting principally of a cleansing solution of boric acid with a weak solution of sulphate of zinc, together with plenty of fresh air, well-regulated diet, and a tonic.

It is a positive mistake to use highly irritant agents such as copper sulphate, silver nitrate, etc., and especially the radical surgical treatment such as is very often employed in the treatment of trachoma. The disease is pactically self-limited and the best results will be had by the use of general cleansing, nonirritating collyria.

CONJUNCTIVAL EPITHELIOMA AT THE LIMBUS

WILLIAM H. CRISP, M.D., OPH.D.

AND

WILLIAM C. FINNOFF, M.D., OPH.D.

DENVER, COLO.

Primary epithelioma of the bulbar conjunctiva, while not of extreme rarity, is yet sufficiently uncommon to render individual cases interesting and significant.

A peculiar feature of this, as well as of other forms of epibulbar growth, is that they may exist and develop over long periods without invading vital structures. Thus in the striking case reported in 1913 to the American Ophthalmological Society by De Schweinitz and Shumway, the growth had probably existed for at least eleven years. The tumor mass measured 27 mm. across and extended backward along the outer surface of the sclera for a distance of 6 mm. on the temporal and 4 mm. on the nasal side; yet the interior of the eyeball was free from invasion, although there was beginning involvement of the perivascular spaces of the perforating ciliary vessels. In this case, the freedom of the interior of the eyeball from invasion may be contrasted with the fact that three months after enucleation the patient returned with an extensive recurrent growth.

Of the sites recorded for carcinoma of the bulbar conjunctiva, the sclerocorneal limbus is perhaps the most frequent. Histologically, the normal eye presents some irregular downgrowths of epithelium in this location. It is commonly stated that penetration of the interior of the eyeball is most likely to occur in those cases of epithelioma developing in this region; largely, in all probability, because of the natural pathway presented by the penetrating ciliary vessels and their perivascular tissue. Nevertheless, the case records show a relatively benign outcome for those cases of epithelioma at the limbus in which the growth has been removed at a fairly early stage of development. This statement applies even to those tumors which are removed early with preservation of the eyeball, although most authors naturally insist on the necessity of deep removal, and subsequent cauterization of the base of the tumor.

In a few cases described, the epithelioma has apparently developed on the basis of an old pterygium, as in the example reported not long ago by Dr. Fagin of Memphis, Tenn.

It is possible that in this relation different writers have varied in their analysis of similar conditions. Thus in 1884 Parinaud described, under the name of dermo-epithelioma, an epithelial tumor penetrated by processes of connective tissue containing numerous vessels, the condition being regarded as benign. But in a case reported by Chaillous, a growth apparently of this character had taken a malignant course. In this instance a flat pterygium-like growth was stated to have developed as the result of a traumatism received forty years earlier, the patient being at the time of record 46 years old. Epithelial tumors of apparently malignant character were removed both from the eyeball and from a corresponding area of the lower lid.

As is commonly true of epithelioma developing elsewhere, epibulbar carcinoma is most likely to appear after the age of 40 years. Yet cases have been recorded as occurring in early childhood.

In the case now reported, the patient was fortunate in coming for examination when the growth was still of small dimensions, and apparently of recent development. The tumor occupied almost the classical location of a pterygium. I am not disposed to believe that it represented a degenerated pterygium, but rather that any small tumor appearing in this location is likely, by virtue of the greatly increased vascularity of the surrounding conjunctiva, to take on more or less of the semblance of a pterygium.

Case 1.—The patient, Mrs. M. J. F., was a woman of 56 years, the wife of a farmer, previously from the middle West, but residing for a year or so past in a small Colorado settlement at an altitude of about 6,000 feet, where wind, dust and sunshine are all extremely abundant. Redness of the nasal side of the right eye had been noticed only for four or five weeks, and the presence of a lump next to the cornea for not more than two weeks, within which time a local general physician had made an incision into the growth, in the belief that it contained fluid. Another physician had diagnosed the condition as pterygium. The patient declared that she had never had any eye trouble before.

The tumor consisted of an irregularly hemispherical whitish mass at the right nasal limbus, its center slightly downward from the horizontal meridian of the eyeball. It measured 4 mm. horizontally by 5 mm. vertically, and protruded about 2 mm. from

the general contour of the eye. It was of gristly consistency. In order to satisfy the patient, I punctured the tumor with a cataract knife, but obtained no fluid, the structure being firm throughout.

Next morning the growth was removed freely under cocain and adrenalin, the base of attachment being curetted thoroughly with a sharp knife, and cauterized with pure nitric acid. There was free bleeding. The growth was most firmly adherent to the eyeball over a very minute area exactly at the limbus. The operative dehiscence in the conjunctiva was filled in by bringing together two sliding flaps from above and below, which were united by two vertical and two horizontal sutures.

I had felt uncertain as to the exact nature of the growth, and therefore submitted it to Dr. W. C. Finnoff of Denver for pathological study. Dr. Finnoff's report was as follows: "Specimen fixed in formalin. Microscopic examination: The surface of the tumor is covered with a thick layer of epithelium which sends columns of cells into the underlying subconjunctival connective tissue. The epithelium near the base is columnar in type, but rapidly changes to polygonal cells, many of which have prickle-like processes. The cells then become flattened and arranged in whorls around a central horny area or epithelial pearl. The epithelial nuclei are typical, many are quite large, many are smaller than normal, while others show evidence of karyokinetic changes. Several extruded nuclei can be seen. In many places these cell columns have penetrated the basement membrane and sent outshoots of epithelial cells into the underlying connective tissue.

"The subepithelial connective tissue is well supplied with blood vessels, many of which are filled with blood. Except for the invasion of new-formed epithelial cells, this tissue is quite normal.

"The presence of atypical epithelial arrangement, with pearls, prickle cells, irregular nuclei and absence of a basement membrane in places, with an invasion of new-formed epithelial cells into the underlying adult type of connective tissue, clearly establishes the microscopic diagnosis of epithelioma."

Recovery was entirely satisfactory, the eye developing a normal scar, of only slightly vascular character. The patient was last seen rather more than two months after the operation, up to which time there had been no sign of recurrence; and was last heard from rather more than four months after the operation, her daughter coming in at that time to report that her mother's eye was perfectly well.

An interval of four months without recurrence does not of course preclude the possibility that this accident may develop at some later date, although judging from the published records of other cases the patient's chance of escape from further trouble in this respect is distinctly favorable.

DISCUSSION

Dr. Joseph Lichtenberg, Kansas City: I did not know I was down for this paper until I saw the program; therefore, in discussing this excellent paper I can give only a cursory review of the literature.

In treating the desiccation method as described by W. M. Clark in a paper before the ophthalmic section of the A. M. A. in Detroit last June and published in *The Jour. Am. Med. Assn.*, July 1, 1916, he says that the desiccation method is peculiarly adaptable to ophthalmic growths, because of the ease of application and great refinement of control.

Zeigler, in discussion, sums up the advantages and disadvantages as follows: Advantages: simplicity of operation, rapid healing without scar tissue, low percentage of recurrences. Disadvantages: The elaborate and costly apparatus necessary to obtain the best results, the danger of too deep destruction of tissue unless the operator is skilful and thoroughly trained in this special field of work.

In 1879 Meyers found that out of 187 epibulbar growths, 17 sprang from the conjunctiva and 129 from the limbus. Epitheliomata form by far the largest number of these growths. It is mentioned by Powers and others that the so-called sarcomatous forms are usually epitheliomata of more rapid growth. True epithelioma of the limbus was reported by Stevast in 1897, most of which started in the conjunctiva, and where the cornea was involved it was only in epithelial layers or the contiguous layers of the substantia propera.

Delord and Revel, in *Annales d'Oculistique*, 1909, cxlii, 432, conclude as follows: Epithelioma of the bulbar conjunctiva usually grows at the expense of the conjunctiva between the cornea and inner angle. Its greatest development is in the region of the limbus, but it can increase and develop around the fibrous ball of the eye. In the latter case the globe resists the penetration of the cancer into the eye, and therefore the epithelioma may be considered relatively benign. It is always proper to be very careful in the exact diagnosis of these lesions and in their prognosis, the unfiltrated part of the limbus having already served as a port of entrance for the cancer. The sclerocorneal limbus is, in fact, the defect in the armor established by the union of the sclera and cornea with each other. It is through this region that epithelioma of the conjunctiva passes from the exterior into the interior of the eye, due to the lymphatic vessels that it controls. The treatment should be abscission followed by Roentgen ray or radium when the cancer rests only on the sclera, but when the limbus is attacked, it is more prudent to resort to enucleation.

According to Saemisch, epithelioma of the epibulbar conjunctiva is most frequent at the limbus, and in the earlier stages there is present a red or gray-red spot resembling a phlyctene, and it is often treated as such. The consistency often varies from the soft and easily dented to the very hard and tough. Sometimes, also, the tumor develops on a very small base, growing to the size of a bean or hazelnut and extruding through the palpebral fissure. Also, at times, these growths are multiple, as in a case reported by Fuchs.

In considering the etiology, the well-known predisposition for epithelioma to occur at places where the epithelium changes its characteristics, as in mucocutaneous junctures, accounts for the greater frequency of the growth occurring at the limbus; also trauma is a factor, the limbus being a region where slight insults are more likely to occur.

The malignancy is not great while the tumor in itself is left undisturbed, but increases greatly when recurrences take place, and the danger of metastasis is also greatly increased. Hence the necessity of thoroughness in the removal.

Dr. Patton, Omaha: I wish to mention a case with a rather unusual etiology which I saw some time ago. The patient, a man of 30, came in with this history: Some two years previously he had gotten some lime in the inner angle of his right eye. There was no previous condition that would suggest an epitheliomatous growth. When seen there was an epithelioma at the limbus about 3/16 of an inch wide and 5/16 of an inch long extending to the bulbar conjunctiva. There was the usual gray roughened surface surrounded by a zone of congestion. The use of radium was suggested, but as he did not want to follow this course, the growth was removed in the usual way, and was found to be firmly attached at the sclerocorneal margin. The base was thoroughly cauterized with the actual cautery, and prompt healing resulted. I have not had a report from the laboratory, but this was the clinical history.

Dr. Robert Fagin, Memphis, Tenn.: My patient, whom Dr. Crisp has just referred to in his paper, and whose right eye I enucleated three years ago, has had no return of an epithelioma, as yet. This was the third successful removal of an epithelioma from this patient's face. The first one came on the nose, the second on the right cheek, and the third on the head of a pterygium at the inner canthus of right eye. Patient is now 79 years old. Six weeks ago he came to see me for an examination of the left eye. I found two pterygia on this eye. I removed the larger one, which was at the outer canthus, and this morning at the Baptist Hospital I removed the other pterygium, which was located at the inner canthus. Neither of these showed any sign of malignancy. Yet I consider the eye safer now than with the growths.

THE PRACTICAL VALUE AND LIMITATIONS OF THE TONOMETER

EDWARD JACKSON, M.D.
DENVER, COLO.

The practical value of an instrument of diagnosis rests on two things: the data that it yields and our understanding of what these data mean. We cannot fully understand what a test is worth without some definite ideas about the limitations of its reliability and usefulness. To find out what the tonometer cannot do, and what false conclusions are likely to be drawn from its indications, is to add to its practical value.

The tonometer of Schiötz measures the depth of an indentation of the eyeball produced by a given weight applied to the center of the cornea through a steel rod. The rod acting on the short arm of a lever moves an index over a millimeter scale. The index being about twenty-five times as long as the arm on which the rod acts, each of its divisions represents an indentation by the rod of 0.04 mm.

As the instrument is applied to the eye the rod is first to come in contact with the cornea, and produces the indentation. But when, with the whole weight resting on it, the displacement of the rod reaches its maximum, the curved base of the instrument also comes in contact with the cornea and begins to bear part of the weight of the instrument. It also indents or changes the form of the corneal surface, without adding to the displacement of the rod. The reading is only accurate when the full weight is borne by the rod and the curved base takes no appreciable part in transmitting the weight to the eyeball.

If the indentation were always of the same shape the displacement of the fluid in the eyeball would be in some way proportioned to the cube of the rod displacement. But the indentation made by the rod varies in shape with its depth, with the intra-ocular pressure, and with curvature, rigidity, and thickness of the cornea. Low intra-ocular pressure, greater thickness, or greater rigidity of the cornea tends to cause a depression that is relatively broad for a given weight. High intra-ocular

pressure or a thin, flexible cornea favors a relatively deep, narrow indentation. In this connection it should be remembered that the corneal center may vary from 0.6 mm. to 0.9 mm. in thickness in normal eyes.

If the corneal plate or base of the tonometer is flatter than the cornea, it comes in contact with the cornea only near its center. Its bearing surface then widens with increase of the indentation. If the surface of the base has the same curve as the cornea, its whole surface is at once in contact and remains so, except as the indentation of the cornea separates the surfaces. Generally the base does not touch the cornea until the rod has been fully displaced and has acted on the index. The reading taken represents this maximum displacement of the rod, the base being just in contact with the cornea. The contact of the base with the cornea, and all the pressure made through it, tends to produce an indentation that gives no indication through the index, but is attended with a relatively diminished displacement of the rod. This undesirable pressure on the globe will have least effect when distributed over the largest surface, and will be least variable when so distributed. It would therefore seem desirable to have a base that corresponds closely in curvature to that of the cornea. But in different eyes the corneal curvature varies enough to be of serious importance. The following table shows the relative frequency of different corneal curvatures among 2,000 eyes, as measured with Javal-Schiötz ophthalmometer:

Corneal Curvatures Among 2,000 Eyes

Radius of Curvature	No. of Eyes	Percentage
Under 6.5 mm.....	6	0.3
6.5 to 7 mm.....	10	0.5
7 to 7.5 mm.....	305	15.25
7.5 to 8 mm.....	1,263	63.15
8 to 8.5 mm.....	409	20.45
8.5 to 9 mm.....	2	0.1
Over 9 mm.....	5	0.25

The Gradle tonometer with its base plate having a radius of curvature of 7.6 mm. fits the cornea accurately enough for all practical purposes in four-fifths of all eyes. But when it comes to dealing with the indivdual eye, averages may be misleading. In the above series of eyes there were five that had a radius of curvature of over 9 mm. On such a cornea, when

the edge of the Gradle base-plate (7.5 mm. in diameter) rests on the cornea the rod drops 0.04 mm. before touching the cornea. A perfectly rigid cornea, which should show by the instrument a reading of 0, would, on account of its flatness, give a reading of 1 on the tonometer scale, corresponding to a difference in supposed intra-ocular tension of 5 mm. to 7 mm., according to the weight used. For the most extreme case of flat cornea encountered in this series, with a radius of curvature of 11 mm., the error would be almost two divisions of the scale, supposed equivalent to 10 mm. or 12 mm. of mercury.

On the other hand, unusual convexity of the corneal surface, other things being equal, causes a greater displacement of index with the same displacement of fluid. The tonometer thus indicates a higher intra-ocular tension than it would on a flatter cornea. This may be of some practical importance, as in the following case:

Case 1.—Miss J. E., aged 31, consulted me to find out if she was beginning to have glaucoma. Her father had suffered from glaucoma secondary to a dislocation of an over-ripe cataract, and her stepmother had primary glaucoma. She had been told she was getting glaucoma, and had been given a solution of eserin, which had caused very severe pain.

The tonometer index registered 8, supposed to indicate an intra-ocular pressure of 32 mm. of mercury, higher than the normal limit has been placed by anyone who has attempted to establish such a limit for tonometric observation. With the ophthalmometer, however, her cornea was found to have a curvature radius of only 7.1 mm. This unusual corneal curvature seemed sufficient to account for the high reading of the tonometer. She was given the opinion that she had no glaucoma and was advised to discontinue her miotic. Three years freedom from any indications of increased intra-ocular tension seem to have justified this view of the case.

The tendency of flatness of the cornea to indicate an indentation greater than exists, or of great corneal curvature to simulate lessened indentation, or increased intra-ocular pressure, can be guarded against by use of the ophthalmometer; although no definite allowance can be made for any given amount of departure of the corneal radius from the average. The ophthalmometer will show also the degree of corneal asymmetry, and the presence of irregularities of surface. In the 2,000 eyes above tabulated, the difference in radius of curvature in the two principal meridians amounted in some cases to more than 2 mm. For such differences, also, it is impossible to apply any

correction to the tonometer readings that would be of practical value. But to know that the surface is irregular and its indentation by the tonometer somewhat anomalous, will guard against the danger of attaching undue importance to its readings.

The effects of certain faults in the application of the instrument to the cornea have been brought out in a paper by Mr. Priestley Smith.[1] He demonstrated that placing the instrument excentrically on the cornea, with axis parallel to the visual axis, has the same effect as relative flatness of the cornea. It tends to an underestimate of the intra-ocular pressure. On the other hand, placing the tonometer rod at the center of the cornea, but inclining the axis of the instrument from the corneal axis, has the same effect as increased corneal curvature (short radius). It brings about an estimate of the intra-ocular pressure that is unduly high. The above are the chief sources of error that lie in the lack of adaptation, or an improper application of the instrument to the eye. The more important and less knowable sources of error lie in the structure of the eye examined.

VARIATIONS DUE TO THE STRUCTURE OF THE EYE

Differences in the rigidity of the cornea make it impossible in any case to know from the reading of the tonometer the actual intra-ocular pressure. A very large number of comparisons with manometric observations upon the dead eye might give us fairly accurate averages. But here, especially, averages are likely to be misleading in the individual case. Priestley Smith's tabulated observations are extremely interesting. He measured the tension of twelve human eyes under known manometric pressures. With an actual pressure of 17.6 mm. of mercury the readings varied from 9.1 to 5.9. That is, the inferred pressure from the readings would, in different eyes, vary from 9 to 16 mm. With 25 mm. pressure the readings varied from 5.1 to 3, and with 39.7 mm. pressure the readings varied from 1.1 to 0, inferred pressures from 35 to 43 mm. He points out that Schiötz' observations were equally variable. Of three eyes with exactly the same intra-ocular pressure one would be recorded as 30 mm., another 36 mm., and a third as 43 mm.

---

1. Ophthalmic Review, **34**, 65.

To illustrate the effect of corneal rigidity, Smith took a fresh pig's eye and tested it under a manometric pressure of 25 mm. of mercury. When perfectly fresh the reading was 3.6, inferred pressure 22.5 mm. When it had been in formalin solution three and one-half hours, the reading was 2, inferred pressure 30 mm., and after twenty-three hours the reading was 0.5, the inferred pressure 61 mm., although the actual intra-ocular pressure was exactly the same, 25 mm. at each test. These facts led Priestley Smith to urge "that when a tonometric observation is recorded it is the reading and not the supposed equivalent in millimeters of mercury that should be stated." The misleading inferences about the limits of normal intra-ocular pressure, and the pressure that should be taken to indicate the presence of glaucoma, have currency and influence through the practice of regarding an inference (often quite an erroneous one) as an accurately observed fact. To keep our records as Schiötz and Priestley Smith advise will serve to constantly remind us where ascertained fact ends and justified inference begins.

But although the tonometer has not taught us the limits of normal intra-ocular pressure, or the boundary of the glaucoma danger zone, it is of great value in showing changes of intra-ocular pressure when successive observations are made on the same eye. Given all other factors constant, the reading of the tonometer corresponds quite closely to the degree of intra-ocular tension. The difference between successive readings of the tonometer given by the same eye at one test should not vary one millimeter, corresponding to a supposed variation in the tension of 2 to 5 mm. With allowance for this amount of possible error, readings on successive days give with great certainty the changes in the intra-ocular pressure of the eye under observation.

CLINICAL SIGNIFICANCE OF TONOMETER READINGS

The most serious limitations on the practical value of the tonometer are due to our scanty knowledge of the clinical and pathologic significance of tonometer readings. When the tension of the eyeball was judged by finger pressure the inexactness of the method could be blamed for uncertainty attending the inferences drawn from our observations. Now that we have a much more accurate means of judging the resistance

of the eyeball to pressure, it must be confessed that much of the uncertainty still remains. It will be a step toward the solution of important clinical problems if we frankly admit that the significance of increased hardness of the eyeball, whether perceived by the fingers or measured by the tonometer, is still very imperfectly understood. It is generally assumed that increased hardness of the eyeball is due to increased intra-ocular pressure. This may be true. But Priestley Smith's experiment with the eye kept in weak formalin solution demonstrates that increased hardness may arise from changes in the sclerocorneal coat, quite apart from any change in the intra-ocular pressure. The hardening of tissue with formalin may be far removed from vital processes, but that pathologic changes may and do increase the rigidity of tissues is a common observation.

In the softer tissues, like the lids, it is always expected that the inflammation will increase the resistance to pressure. There is reason to suppose that the same thing occurs in the sclerocorneal coat. The diminished indentation of the eyeball by the tonometer depends not merely on increased rigidity of the cornea. Indentation is brought about by displacement of fluid in the anterior chamber. This is possible only as the escape of fluid from the eyeball or the distensibility of the corneoscleral coat makes room for it. Increased resistance of any large part of the coat must increase the hardness of the eyeball, all other things remaining unchanged. It is probable that inflammation involving the corneo-scleral coat causes such increased rigidity of it as to produce noticeable increase in the hardness of the eyeball, without any actual increase of intra-ocular pressure on the part of the contents of the eyeball.

Fischer's theory of edema[2] applied to glaucoma has been the subject of experiment by himself, Ruben,[3] and McCaw[4]. All agree that the excised eyeball placed in a weakly acid solution becomes extremely hard, often bursting the sclera. Fischer ascribed the hardening to increased intra-ocular tension; Ruben to thickening and diminished capacity of the sclerocorneal coat. He found that the coat increased in weight during the process as much as 245 per cent. Such thickening was present in McCaw's experiments and the change in the

2. Hatfield Prize Essay, College of Physicians of Philadelphia, 1909.
3. Graefe's Arch. f. Ophth., **86**, 258.
4. Ophth. Rec., **24**, 284.

sclerocorneal coat, quite apart from the pressure of its contents, may produce increased hardening.

The following experiments were made with Dr. McCaw's assistance:

Sheep's eyes were tested under known manometric pressure first when fresh, then after exposure for one day to acid solution. At the second test all showed thickening with softening of the corneoscleral coat. In some the intra-ocular pressure was increased, in others diminished; but in all it was higher than it would have been in eyes kept in plain water. In this case average results were significant. These were as follows, the inferred pressure in millimeters of mercury in parentheses:

Manometric Pressure, 37 mm. Hg	75 mm. Hg
Fresh eyes .....10.7 (21. mm. Hg)	4.4 (50 mm. Hg)
Thickened coats ..... 8.0 (32 mm. Hg)	4.7 (49 mm. Hg)

The average of reading before the fresh eyes were connected with the manometer was 13.2, inferred pressure 13 mm. Hg. The sheep's eye has a relatively flat cornea.

For the lower intra-ocular pressures the tonometer reading was distinctly higher in the eyes after the corneoscleral coat had been thickened; while with the higher intra-ocular pressure the effect of thickening the corneoscleral coat was very slight.

Gross changes in the corneo-scleral coat, whether of curvature or consistency, destroy the value of tonometric readings. This has been generally recognized. It is more important to recognize that great diminution in the impressibility of the cornea may arise without evident change in this coat, and without indicating the clinical entity or syndrome, known as glaucoma. If we speak of these conditions as constituting "temporary glaucoma" we give the word glaucoma to a mere symptom, without special etiology, pathology, prognosis, or treatment.

It is the most important limitation on the practical usefulness of the tonometer, that it gives readings from which high intra-ocular tension and glaucoma would be inferred, when the disease glaucoma, or any special tendency to it, is entirely absent.

This is generally understood with reference to rise of tension following discission of the crystalline lens, as in the following cases:

Case 2.—Miss M. D., aged 20, was examined and her eye normally gave a tonometric reading of 9 to 10 with Gradle's modification of the Schiötz tonometer, inferred pressure of

24 to 28 mm. A few hours after free discission of the lens nucleus the reading was 3, inferred pressure of 57 mm. This dropped back to normal next day.

Case 3.—In the case of Miss A. H., aged 18, the tonometric readings after a needle operation varied from 7 to 5 (37 mm. to 47 mm.) for about four weeks, although her normal reading was 11, inferred pressure 20 mm.

A similar rise of tension may follow operations for secondary cataract. A week after such an operation the tonometer registered 4, 52 mm. inferred pressure, but subsequently became and remained normal, 9, inferred pressure 28 mm. After discission in young people glaucoma would rarely be thought of. But, as it is known to follow operations for secondary cataract in elderly people, high tension persisting for a week might very readily lead to unnecessary and harmful treatment. The following case was still more puzzling, because a dislocated lens is one of the important causes of secondary glaucoma:

Case 4.—Mrs. A. R. J., aged 70, had had double senile cataract, and the left lens had been extracted twelve years before. She came with a hypermature Morgagnian cataract of the right eye, the lens nucleus, about 5 mm. in diameter, having fallen until its upper margin showed in the lower edge of the pupil. A week later the eye developed pericorneal redness, pain, and increased tension. The Gradle-Schiötz tonometer reading was 4, inferred pressure 52 mm. Pilocarpin contracted the pupil, but gave no relief. Homatropin was then used, and in two days the tonometric reading was changed to 7, inferred pressure 37 mm. She was put on atropin and two weeks later the pupil was well dilated, the eye was nearly free from hyperemia and comfortable, tonometer reading 8, inferred pressure 32 mm. Five weeks later, at the time the atropin was discontinued, the eye was entirely quiet, the tonometric reading 9, inferred pressure 28 mm., and two months later it was in this same condition.

We must recognize that hyperemia or inflammation of the uveal tract is likely to be attended by increased intra-ocular pressure quite apart from the disease we know as glaucoma.

Case 5.—Miss S. L. C., aged 51, had been subject to attacks of uveal and corneal inflammation from childhood. On the fourth day of a severe attack of uveitis her eye was found to give a tonometric reading of 5 to 6, inferred pressure 42 to 47 mm. There was great pain, but with active treatment for iridocyclitis, the tension fell so that two days later the reading was 10, inferred pressure 24 mm., and under atropin it fell in one week to reading 14, inferred pressure 11 mm.

In this case only the experience of similar attacks in the same patient prevented a mistaken diagnosis and wrong treatment. Four years have elapsed without any other evidence of glaucoma. The tension of this eye is generally as when last taken with reading of 13, inferred pressure 14 mm.

Case 6.—Shortly after encountering the above case, Mrs. H. E. W., aged 70, came with pericorneal hyperemia and pain in the eye. The pupil had dilated freely (6½ mm.) under homatropin, when it was found that the tonometric reading was 4, inferred pressure 52 mm. Because of the clinical evidences, and the experience with the case above mentioned, this patient was put upon atropin. Three days later the tension had diminished so that the tonometer read 6, inferred pressure 43 mm.; three days after this the tonometer reading was 8, inferred pressure 32 mm.; and two days later tonometer reading 10, inferred pressure 24 mm. This patient recovered completely from the iritis, without adhesions or exudates to impair vision. Three years have passed without recurrence of iritis or any symptom of glaucoma.

Even choroidal disease, without evidence of involvement of iris or ciliary body, may be attended with hardening of the eyeball.

Case 7.—Miss G. N. H., aged 23, had come to Denver for tuberculous disease of the choroid involving the region of the right macula, three years before. This had been quiet for the eighteen months that she had been under observation, when she noticed extensions of the scotoma; and the ophthalmoscope showed recurrence of the disease at the margin of the former patch. On the fifth day of the attack the right eye became painful with some general hyperemia, and she noticed rings of color, the diffraction spectrum, about the light. Tension with figures was recorded as plus 1. With the Gradle-Schiötz tonometer the reading of the right eye was 2 to 3, inferred pressure 60 mm. Left eye 11, inferred pressure 20 mm. Pilocarpin was instilled in the right eye and later 0.1 per cent. solution of eserin repeated within one-half hour. Next day the pupil was well contracted and the eye less painful, but the tension continued high. Six days after increased tension was first noticed the reading was 3 to 4, inferred pressure 55 mm.

The eserin solution was continued and ten days later tonometric reading for the right eye was 11, inferred pressure 20 mm. A month after this the reading went to 9, inferred pressure 28 mm, but quickly dropped to what seemed to be the normal reading of 11 for each eye. Under tuberculin injections and attention to general health the process in the choroid became entirely arrested, leaving her with a larger patch of atrophy in the macula and a corresponding scotoma. Four years have

elapsed since the last evidence of increased tension, and the eye, except for the macular lesion, seems quite normal.

Several other cases more or less resembling these instances of uveal disease have been encountered. They have not been so long under observation, but in none of them was there any evidence of glaucoma subsequent to the cure of the uveitis.

In conclusion, it should be emphasized that the tonometer of Schiötz, or the convenient modification of it by Gradle, is an instrument of great practical and scientific value.

A single test with it cannot be relied on as indicating with any certainty the intra-ocular pressure.

A comparative test with the two eyes, when one shows nearly the average reading for a normal eye, and the other a marked departure from it, raises a strong presumption of departure from the normal intra-ocular tension in the latter eye.

Repeated tests of the same eye giving differences in the tonometric reading indicate with certainty pathologic change, generally a change in the intra-ocular tension, so that the instrument gives very accurate indications of the course of the case.

Far from diminishing the usefulness of the tonometer, the readings obtained in uveitis indicate a larger field for its application in which it will be a very valuable assistance. It seems certain that diminished intra-ocular tension is not a constant or pathognomonic symptom for all forms and stages of uveitis.

DISCUSSION

Dr. Harry Gradle, Chicago: We are deeply indebted to Dr. Jackson for this paper. We have gotten too far into the habit of talking intra-ocular tension and inferring that that means something. It simply means we have a measurement that we record in millimeters of mercury. This is not absolute intra-ocular tension and means nothing unless it be excessive. Of course, it stands to reason that the reading obtained with corneae of varying thickness, with rigidity of the sclera, and with the various other conditions we find in an eye, translated into millimeters of mercury, will not correspond with the absolute intra-ocular tension.

The value of tonometry rests on comparative readings: the comparison of the tension of one eye with the tension of the other, the comparison of several readings of the same eye taken several days apart. If I may take your time for a moment, I should like to quote from an article that I wrote some two years ago:

"We are forced to judge of the value of our therapeutic measures from three angles. First, the curve of intra-ocular pressure tonometrically registered. A curve should be plotted for every patient with readings made at least twice a week. The course of this curve is a fair indication of the immediate value of the miosis induced. Second, examination of the patency of the intra-ocular outlets. At the first examination of the patient the tension of the eye should be measured before and after three minutes of deep massage of the eyeball, and this should be repeated every

two weeks. If the intra-ocular outlets are patent to a minor degree, or if they possess the potentiality of being opened, we have an indication from the massage that the disease may yield to conservative treatment. But if no reduction of hypertension, or at the most a very slight reduction, follows the massage, we are justified in assuming that operative interference will be necessary to re-introduce the intra-ocular pressure balance. More or less empirically speaking, a temporary reduction of at least four to six millimeters of mercury should result from three minutes of deep massage to prove the potential patency of the outlets.

"I do not wish to speak of tonometry at length, but do wish to emphasize one point, the disregard of which causes the majority of errors that occur in the use of the instrument: The anteroposterior axis of the eye must be absolutely vertical and the tonometer must be so placed as to be a direct continuation of this axis."

We should use two weights in making our readings, first one weight and then the added weight, and in case of necessity with a third weight, in order that we may know whether the instrument is in the right position upon the cornea. These weights are simply to check up our readings and induce greater accuracy. Again the absolute tonometric reading is of practically no value at all. It is the comparison of the tonometric reading of one eye with the other eye, the tonometric reading of one eye of one date with that of some previous or future date that gives us the value of the tonometry and the value of the data thus obtained is inestimable.

Dr. Wendell Reber, Philadelphia: One point that I strained both of my ears to hear Dr. Jackson's paper discuss, and could not hear with either ear, was: What the normal tension is? I hope that in closing the discussion Dr. Jackson will give us his valuable idea as to what the normal tension is. We used to consider it from 18 to 23. My own feeling is that it runs from 25 to 30. Any patient will give a more intelligent reading at the second examination than at the first, because of mental excitement over what is being done. The increased tension of uveitis is an illustrious example of the iritis of glaucoma mentioned twenty years ago. I, myself, have had trouble with two such cases, in which it was difficult to say whether the case would prove to be uveitis or glaucoma. It was one of those borderland cases in which I believe the tonometric study will be of value. We hear practically nothing about lowered tension, the lowered tension of iritis and uveitis. Everybody is ready to study increased tension, but nobody seems ready to study decreased tension. We should study both. Of course, the thickness of the tunics influences the reading. The ideal instrument is the one-man instrument, although almost every instrument is a two-man instrument. The Schiotz and the Gradle are two-men instruments. The Stevenson has the same idea in view. Souter of Boston has one.

Dr. Edward Jackson (closing discussion): I have very little to add. I think the question of normal intra-ocular pressure is not as yet settled. Priestley Smith, in this article that I referred to, remarks that it is hard to concede that there should be such variations of normal pressure as have been supposed to exist. He believes the normal pressure is more nearly constant than has generally been supposed. The widest range was from 12 to 30 mm. My own impression is that if the tonometer be relied on to give the normal intra-ocular pressure, it would be from 25 to 30 mm. I may confine my remarks to the Schiötz instrument; yet I feel that it is not the ultimate goal of tonometers. It is not the best tonometer that is possible. It is quite possible the solution will be something like the Souter tonometer, or something like the arrangement that Dr. Coburn

of Chicago proposed some years ago, whereby the amount of displacement of fluid was pretty accurately measured, and the pressure was measured which was required to produce a constant displacement in the eye. Something in that direction may give us a more valuable instrument than we yet have. I can conceive of the Souter tonometer being made more accurate, perhaps, by viewing the reflection from the corneal surface under a magnification. In that way we could study the depression that is made, and if we could study the different forms of depression, we should probably arrive at a better understanding of the meaning of the depression.

CONJUNCTIVITIS TULARENSIS (SQUIRREL-PLAGUE CONJUNCTIVITIS)

FREDERICK W. LAMB, M.D.
CINCINNATI, OHIO

I wish to present for consideration and discussion, an unusual case which recently came under my observation, the third case to be reported of what seems to be a new disease in ophthalmology.

The first case on record was reported by D. T. Vail, in a paper read before the Michigan State Medical Society Sept. 10, 1914, and published in the *Ophthalmic Record*, October, 1914. This case was also reported by Wherry and Lamb in the *Journal of Infectious Diseases*, September, 1914.

The second case was reported by Robert Sattler, before the American Opthalmological Society in 1915, and published in the *Archives of Ophthalmology*, May, 1915.

The third case is the one I wish to report today.

CASE 1.—C. F., a young colored girl, was first seen by me on Nov. 16, 1916. She complained of pain and swelling in the left eye, accompanied by some discharge. The eye had been steadily growing worse since the beginning of the trouble, three days previous, and the day before I saw her she had a chill, followed by headache and a general feeling of malaise.

There was edema of the eyelids and a watery discharge. The conjunctiva of the lower lid was the site of six or seven small sharply defined deep ulcers, filled with yellow necrotic plugs. The conjunctiva of the upper lid was free from ulcers at this time. The pre-auricular gland on the left side was very much swollen and tender, the cervical glands were tender but not much enlarged. Questioning brought out the fact that two days before the onset of her trouble she had prepared some rabbits for the evening meal of the family by which she was employed. A diagnosis of conjunctivitis tularensis (or squirrel-plague conjunctivitis) was made, and she was sent to my service in the Cincinnati General Hospital. Dr. Wherry and my brother, Dr. Benjamin Lamb, studied the case bacteriologically and confirmed the diagnosis.

Guinea-pigs inoculated with secretion from the eye died on the fourth day, and from the numerous miliary abscesses in the spleen and liver, the bacillus tularensis was isolated. Inoculations of guinea-pigs with blood from the patient were negative. Inoculation of ordinary culture media showed no growth.

Dr. Benjamin Lamb was able, however, to grow the bacillus on egg yolk medium from inoculations made directly from the conjunctival ulcers. He also made an autogenous vaccine which we used therapeutically with most gratifying results.

On November 20, four days after I first saw her, and eight days after the onset, the disease had extended so that the discrete ulcers had fused into three or four large ones, and four small ulcers had appeared on the conjunctiva of the upper lid, two near the upper margin of the tarsus and two near the free border of the lid. The swelling of the lids had been reduced considerably by cold applications and flushing the conjunctival sac with boracic acid solution. The temperature of the patient was somewhat septic in type, varying from 101 to 103, pulse 90 to 100. Her mental condition was drowsy and stupid. She complained of very little pain in the eye, but the glands of the neck were exquisitely tender.

On November 24, she received one-half minim of vaccine subcutaneously in the left arm. There was no systemic reaction, the highest temperature on the following day being 101, but at the site of the injection there appeared several blebs surrounded by an indurated area, the size of a dollar. The following day (Nov. 26) the temperature rose to 102. The appearance of the eye was about the same.

On November 29, five days after the first injection of vaccine, she received one minim in the right arm. There was a slight systemic reaction following this injection, the temperature going to 101, accompanied by headache, "stiff neck," and pain in the head and shoulder. The local reaction was much like that following the first injection.

The next day there was no rise of temperature and the appearance of the eye was much better. There was less secretion and the necrotic tissue had sloughed away from the ulcers, leaving clean-looking, bright red granular bases, rather deep but clean. Her mental condition was much better, and she asked permission to be out of bed. Her temperature now remained normal until December 4, when she was given another minim of vaccine in the left arm. On December 5, her temperature rose to 102, pulse 120, with some headache and pain in the arm. On December 6, temperature and pulse were again normal. The swelling had nearly all disappeared from the lids. The small ulcers on the upper lid had become much smaller and the larger ones on the lower lid much shallower. The pre-auricular gland was still swollen and tender.

On December 10, the ulcers of the upper lid had disappeared and those on the lower lid had become much smaller. Recovery is now assured.

The following bacteriologic examinations were made by Drs. Wm. B. Wherry and Ben H. Lamb:

"On November 16, pus from the eye was inoculated into the peritoneal cavity of a guinea-pig. The pig died in three and one-

Conjunctivitis tularensis
(Squirrel-plague Conjunctivitis)
Frederick W. Lamb

half days with typical lesions—caseous necrosis of the abdominal muscles at the site of inoculation; marked subcutaneous edema; a viscous peritoneal exudate and numerous foci of necrosis in the liver and spleen. *B. tularensis* was isolated from the spleen on coagulated egg-yolk under aerobic and partial tension conditions, but there was no growth under anaerobic conditions on this medium. Attempts to cultivate it on a number of other special media containing human blood or ascites fluid failed.

We have always failed to find the bacterium in smears from human cases, but direct cultures from the eye made on egg-yolk slants on November 20, yielded quite a few colonies of *B. tularensis* along with those of *B. xerosis*. A vaccine was prepared by suspending the growth from a 48-hour culture (without scraping off any of the egg-yolk) in sterile 0.85 per cent. salt solution. This was heated to 55 C. for 45 minutes, cooled, and preserved with 0.5 per cent. phenol. The suspension was so dilute that it hardly exhibited any opacity. When the phenol was added to the heated vaccine it immediately became viscous, like solution of salicylic acid. One-half minim of this vaccine inoculated subcutaneously into the patient's arm produced over night a marked edematous swelling about 3 by 4 inches, and this was covered by vesicular blebs containing clear serum. At the same time the temperature rose 1 degree F. There were no other constitutional symptoms. A similar reaction was produced by each successive inoculation. This simulates the mallein test for glanders and suggests the use of such a vaccine in the diagnosis of obscure cases or in cases which have progressed too far for bacteriologic diagnosis. Late in the disease, though active symptoms are present, it may not be possible to demonstrate the bacilli. On December 26, over a month after this case came under observation, the pre-auricular gland was greatly enlarged (about 1 by ¾ inch) and soft. It was opened and the pus was cultured and inoculated into a guinea-pig without results."

So far as I have been able to ascertain, this is the third case to be reported of infection of the conjunctiva with bacillus tularensis. This bacillus was discovered and first described by McCoy and Chapin in 1911, during their work in California on bubonic plague.

They studied a plague-like disease occurring in California ground squirrels and isolated a bacillus which they first supposed to be identical with the cause of bubonic plague in man, the bacillus pestis. Further study, however, revealed points of differentiation, and they called the disease in squirrels the squirrel-plague, and the bacillus which caused it the *Bacillus tularensis,* from Tulare County, California, where it was then prevalent.

The disease is fatal in squirrels, rabbits, guinea-pigs, and other rodents. Infected guinea-pigs always die in from four to six days.

Two years ago, during the autumn hunting season, it was noticed that wild rabbits were dying in large numbers in the country districts around Cincinnati. Some of these dead rabbits were submitted to Dr. Wherry, and from them he isolated the *Bacillus tularensis*. The two cases reported previous to my case occurred at this time in Cincinnati, and the history developed the fact that both patients had handled dead rabbits.

The infection is undoubtedly transmitted by direct contact with infected material. In all of the cases so far reported it was dead rabbits. A peculiar coincidence is that all three of the cases have had the infection in the left eye.

The progress of the disease in the cases previously reported has been very slow and tedious, one of the cases having passed from observation before the eye was well and the other remaining in the hospital for two months.

In my patient who has now practically recovered at the end of the fourth week, I am convinced that the use of the vaccine has greatly shortened the time of recovery. There was marked improvement after the first dose and normal temperature after the second, with rapid improvement in the appearance of the eye.

The disease seems to be a new one in ophthalmology, although it is possible that unrecognized cases have been seen by men who have not had the advantage of collaborating with a bacteriologist familiar with the work of McCoy and Chapin.

Further Notes.—On December 10, the patient received another injection of vaccine, following which there was some local reaction and a rise of temperature to 101. The pre-auricular gland became more swollen, painful, and tender. On December 26, this was incised and about 10 c.c. of gelatinous fluid and pus was evacuated. Inoculation of guinea-pigs with this material proved negative. It is interesting to note that after each injection of vaccine the swelling and tenderness of the pre-auricular gland increased, but the appearance of the eye improved.

On Jan. 1, 1917, the eye was entirely healed.

GLAUCOMA SIMPLEX WITHOUT PERCEPTIBLE RISE IN TENSION

HARRY S. GRADLE, M.D.
CHICAGO

Under this title, I wish to bring up for reconsideration a definite type of noninflammatory glaucoma. Von Graefe recognized the disease, but did not class it as a glaucoma (Amaurose mit Sehnerven Exkavation), and it remained for Schmidt-Rimpler, some seven years later, to classify it as such. Owing to the difficulty in diagnosis and the rarity of the condition, reports of this type of glaucoma occur but seldom. I refer to the class of cases known as "Glaucoma simplex without perceptible rise in tension."

Sattler claimed that these cases invariably must at some time have an increased tension and I am reopening this subject because I have seen recently a case of this character during one of the periods of increased tension.

CASE 1.—A single woman, aged 67, was brought to me in consultation because of an increase in her myopia and a decrease in her vision. Four years previously, —0.50 sphere, combined with a weak cylinder, gave her R. E. $^{15}/_{30}$ and L. E. $^{15}/_{20}$. The sphere has been increased on three different occasions until she now wears: R. E. — 1.00 s with cylinder and L. E. — 1.50 s with cylinder, giving her a vision of $^{20}/_{80}$ and $^{20}/_{30}$. She has had some headaches. Several years ago, her left ethmoid was exenterated for cerebral rhinorrhoea of several years duration. This subsided after some months and has given her no trouble for over a year. Today she requires a —2.50 s and —2.00 s R. E. and L. E. to obtain V. of $^{20}/_{80}$ and $^{20}/_{30}$.

Externally both eyes are normal and the pupils react well. The right lens and vitreous are clear. The peripheral retina appears normal, but the peripapillar retina is slightly hazy and fluffy, although no exudate could be seen. All of the vessels are slightly dilated, but not tortuous. The disk edges are blurred in spots, as though covered by an edematous translucent retina. The disk is of a peculiar gray color with darker shadows near the periphery. There is positively no glaucomatous excavation.

The left eye shows a very similar condition, with the exception that the edges of the disk are clearer and the gray color of the disk more pronounced. There is no marked difference in the niveau of the disk and the surrounding retina in either eye.

The blind spots are very slightly enlarged, if at all, but not pathologically. No scotomata can be found. The fields are practically normal. The tension of the right eye was 30 mm. Hg, which was reduced by one minute of massage to 20 mm. Hg. The tension of the left eye was 24 mm. Hg.

Because of the increase in the myopia, the headaches, the inexplainable decrease in vision, and the ophthalmoscopic appearance of the disk, I concluded that this was one of those cases of "Glaucoma simplex without perceptible rise in tension." Moreover, the increased tension in the right eye, which yielded quickly to massage, confirmed this view, provided we accept the before mentioned statement of Sattler. The mental disturbance occasioned by a long railroad voyage to the city and the uncertainty regarding her condition were probably the exciting factors of this passing rise of pressure which had never before been noted. Her ophthalmologist had tested her tension several times, although only with his fingers. Since her return home to a quiet life, the tension has again sunken to less than 30 mm. (during weekly readings).

This disease seems to be confined to individuals over 60 years of age and is always bilateral, although one eye is usually more involved than the other. A low degree of myopia, with or without astigmatism, is usually present, and it is the increase in this myopia that often brings the patient to the ophthalmologist. Occasional headaches are the rule, but they are not of the unrelievable type accompanying inflammatory glaucoma. Apparently, this symptom is the result of the infrequent and usually overlooked temporary rise in tension before alluded to. Externally, the eyes appear normal. Until late in the disease, the pupils react freely and normally, although in the later stages, the reaction to light becomes slower and eventually disappears; but the characteristic dilatated pupil of the ordinary glaucoma is entirely lacking. Both macroscopically and microscopically, the chamber angles are free from adhesions. The lens may show a few striated opacities; but I consider them to be incidental rather than the result of the disease. During the earlier stages, the optic nerve heads show but few changes. When seen during a period of increased tension, a slight edema of the retina, simulating a low-grade neuritis, may be observed. But while the tension remains within the so-called normal limits, the only ophthalmoscopic changes to be found consist in a gradually increasing area of pigment and chorioidal

atrophy surrounding the disk and a gradually increasing pallor of the disk. This peculiar color is not at all like that of either ascending or descending atrophy, but is ash-gray. Near the edges of the disk is a darker area, as though a shadow were cast here. The floor of the disk is not markedly depressed nor is there any glaucomatous excavation. The vessels may be slightly dilatated, but show no marked changes. The peripheral retina is normal. If seen during a period of increased tension, there may be a fluffiness of the retina immediately around the disk, but this does not extend into the periphery, and soon subsides.

As the disease progresses, the atrophy of the nerve head advances and the typical glaucomatous cupping makes its appearance. Now the tips of the lamina cribrosa begin to show and henceforth are constantly to be seen. The cupping is very uniform, in contradistinction to the other types of glaucoma in which the cupping usually appears at one side of the disk and spreads around the periphery. Furthermore, the process is very slow in its development, requiring three years in one case that I observed. During this time, the vision gradually decreased, irrespective of the increase in the myopia and of the fairly well-preserved visual fields.

As these patients are seldom under constant observation, the curve of intro-ocular pressure has never been studied accurately. Practically all observers agree that the tension seldom exceeds the limits empirically called normal, i. e., 24-26 mm. Hg. But there is no doubt in my mind that at times there is an increase in the tension. This accounts for the irregular headaches. However, we do not see the marked increase in tension so characteristic of the other forms of glaucoma. The visual fields show but few changes. There may be some concentric narrowing, especially for colors; but the marked defects, both peripheral and pericentral (Bjerrum) are entrely lacking. This remains true, even in the later stages of the disease.

The prognosis must be considered fairly good, because the disease does not appear until late in life and then is very slow in its development. After a complete excavation of the nerve head has appeared, the progress seems to be even slower than during the earlier stages. As a rule, the patient succumbs to some intercurrent trouble before blindness intervenes.

A case well illustrating this was:

Case 2.—An old man, aged 68, consulted me in May, 1911. He complained of diminution of vision during the past year and of "black cobwebs" before the right eye. A —0.50 sphere combined with a weak cylinder gave him bilateral vision of 0.9. Externally both eyes were normal and the pupils reacted normally. The anterior chambers were of normal depth. A few faint lenticular opacities and one small vitreous floater in the right eye accounted for the "cobwebs." Under mydriasis, I found a myopic crescent and peripapillar atrophy, such as is frequently seen in normal myopic eyes. A central excavation of the nerve head proved to be physiologic and not glaucomatous, in that it was not very shallow and did not extend to the margins of the disk. The tension on palpation was normal.

One year later (April, 1912), the condition was unchanged except that the vitreous haziness in the right eye had disappeared.

In July, 1915, his corrected vision was R. E. 0.6 and L. E. 0.8. The ophthalmoscope gave the same findings.

In June, 1914, the corrected vision was R. E. 0.5 and L. E. 0.8. The pupils reacted well. Ophthalmoscopically, it looked like a distinct glaucomatous excavation within a myopic crescent. (Right = Left). Tension was normal. Fields were practically normal. I prescribed pilocarpin.

April, 1915, pupils react well. Both disks show positive shallow glaucomatous excavations. Tension is normal. Fields are slightly contracted concentrically, right more than left.

June 1916, the corrected vision was: R. E. 0.4 and L. E. 0.3. The blood pressure was 210. The tension was normal. Large peripapillar choroidal atrophy and marked increase in depth of excavation whose walls are now nearly straight up and down, right to a greater extent than left. Relative paracentral scotoma.

July, 1916, Exitus from carcinoma of prostate with metastases.

Treatment is of but little avail. Mental quiet and avoidance of extreme physical exertion are to be recommended here as in other conditions. An increase in the blood pressure does not seem to have any marked influence on the course of the disease. Miotics are naturally to be resorted to and I prefer a weak solution of pilocarpin. Complete miosis is not of any greater value than incomplete and the discomfort it entails on the patient reacts unfavorably. Bietti warmly advocates the use of a strong eserin solution and claims that the patients soon become accustomed to it. I cannot recommend that course of procedure. On the other hand, diagnostic dilation of the pupil does not superinduce an acute rise in tension with its accom-

panying disturbances, as it will in the other forms of glaucoma; but it is best to avoid any possibility of danger from this source. Under no circumstances is operative interference to be resorted to.

How must these cases be regarded? As a disease affecting the exchange of fluids in an eyeball, so predisposed that the upper limits of normal tension lie presumably below 20 mm. Hg (cupping a result of pressure); or as a disease of the optic nerve, resulting in an excavation of the nerve head such as studied by Schnabel and Elschnig, without any marked disturbance in the exchange of intra-ocular fluids (cupping not the result of pressure); or as a combination of these two purely speculative types?

Stock observed a case of typical glaucomatous excavation in a 60 year old woman, whose visual fields were so contracted as to include completely the point of central fixation in one eye, and up to within 10 degrees of this point in the other eye. The tension, taken in the hospital at frequent intervals during the period of several days and nights, never rose above 23 mm. Hg. Even after a LaGrange sclerecto-iridectomy, the tension remained around 17 mm. Hg. Stock concluded that this was a case wherein the upper limits of the normal tension lay under 20 mm. Hg. Fuchs concurs in this view and states that "there are cases of glaucoma simplex in which the tension is never found distinctly increased." Furthermore, "in these cases, we must assume that the lamina cribrosa is particularly yielding so that it is forced backwards by a pressure that does not perceptibly rise above the normal limits." In support of the latter view, he argued that if such cases are dependent on the structural differences of the lamina cribrosa (and the different degrees of development of the lamina speak for this), then the reverse must occur; i. e., in spite of long-continued high tension, if the lamina is extraordinarily well developed, an excavation will appear only very late. He recited the details of such a case and Markbreiter also described a similar one.

Orr reported a case of simple glaucoma with cupping of the disks without perceptible rise in tension and explained it by rises of pressure so slight as to be within the normal limits for the average eye. Wicherkiewicz blames an abnormal rigidity of the sclera in these cases, while Sym lays the fault to an unduly low resistance of the nerve head.

These authors thus endeavor to explain the cases of glaucoma simplex without perceptible rise in tension by the view that, in certain eyes predisposed toward glaucoma, there occurs at irregular and unstated intervals a rise in tension slightly above the limits usually accepted as normal. Furthermore, these eyes are of such a type that moderate intra-ocular pressure is pathological, although for the average eye, the same pressure would fall well under the limits of the normal. The generally accepted explanation for this lies in an underdevelopment of the lamina cribrosa which is forced to recede.

This theory cannot explain some of the clinical or microscopical features observed and described. A shallow anterior chamber with even partial obliteration of the chamber angle or with angle adhesions, has never been reported. In other words, the canal of Schlemm is patent and no obstruction to the outflow of the aqueous can be seen. This may be due to the comparatively small number of cases that have come to pathological examination. In one of the few cases thoroughly examined, Elschnig found the anterior segment of the eye normal and the lamina cribrosa in its normal position. Yet this was an undoubted case of glaucoma simplex without perceptible rise in tension. Furthermore, if the excavation were the result of pressure, relief of the relative hypertension should put a stop to the advance of the cupping. But Schmidt-Rimpler merely voiced the experience of all ophthalmologists in saying: "It occurs that the trouble in the nerve (cupping) may progress rapidly after the pressure has been relieved by operation." Acceptance of this view alone places too great a strain on the imagination.

The second view is the outcome of histologic examinations by Schnabel and Elschnig of glaucomatous nerve heads. The former reported the presence of cavernous-like spaces within the head of the optic nerve, as well as behind the lamina cribrosa, in cases of glaucoma simplex. These empty spaces represent an absorption of the individual nerve fibers. In simple atrophy, a similar absorption occurs, but the spaces thus caused are filled out with neuroglia, while in the glaucomatous nerves, no new neurogliar tissue can be found. Ichikawa corroborated the presence of these caverns in five out of ten glaucomatous eyes examined and concluded that the changes occurred first in that portion of the nerve bearing the retinal

vessels. Stock agreed as to the presence of these caverns, but claimed them not to be pathognomonic for glaucoma. He reported finding similar caverns in five cases of high myopia. But the myopic caverns are essentially different from the glaucomatous.

With this histologic picture before him, Schnabel advanced the view that the cupping in glaucoma simplex is the result of a glaucomatous disease of the optic nerve and absolutely independent of the intra-ocular pressure. The nature of this disease could not be determined, but it was probably of the character of a specific noninflammatory neuritis, resulting in a breaking down and eventual absorption of the individual nerve fibers, particularly in front of the lamina cribrosa. Lauber agreed with this view in his remarks about a case of glaucomatous cupping that extended to the edges of the disk and was accompanied by a tension of only 18 mm. Hg. However, this view is not satisfactory for all of the details of the disease.

How, then, can we explain on purely theoretical grounds these cases of glaucoma simplex without perceptible rise in tension? Both schools unquestionably are right in certain points, but I believe that the explanation lies along somewhat different lines than either school has elaborated on; to wit:

(1) The cases now classed as glaucoma simplex without perceptible rise in tension belong, I believe, to a disease *sui generis*, that is, neither a true glaucoma nor a true uveitis. My justification for this statement lies in the fact that the only points this disease has in common with glaucoma simplex are an excavation of the nerve head and an occasional rise in tension, so slight that it might well be within the normal limits. The cases reported by Fuchs, Bietti, Lauber, Elschnig, and others, as well as my own, prove these points.

(2) The disease arises as a low-grade neuritis, limited to the anterior and vessel bearing portions of the optic nerve and leading to an absorption of the nerve fibers that produce the caverns described by Schnabel. The gradual advance of this process results in the so-called glaucomatous excavation of the optic nerve head, but requires many years for its completion. I cannot believe that this cupping is the result of pressure, for were it sufficient to produce that result (even granting a poorly developed lamina cribrosa), we should find at least a

shallow anterior chamber, if not partial obliteration of the chamber angle. But both clinical and histologic reports all show a normal chamber angle and patent canal of Schlemm. Furthermore, a tension sufficient to result in pressure cupping would certainly cause a pressure atrophy of the peripheral retina, which is not the case here.

(3 The intermittent rises in tension are probably not the result of interference with the outflow of the intra-ocular fluids, but rather an increase in the secretion of these fluids. We know from the masterly work of Leber and latterly of Troncoso that the aqueous (which, after all, is the balance of intra-ocular pressure) is secreted at the rate of about 5 to 6 c.mm. per minute, and that is probably all that the flaccid lymph channels of Schlemm can take care of. Thus any marked increase in the rate of secretion would tend to carry the intra-ocular tension toward the upper limits of the normal and possibly above.

What, then, is the cause of this increased secretion? In a case of this kind reported by Elschnig, there was found in addition to the glaucomatous process in the optic nerve, an extremely low grade of chronic uveitis, probably due to the absorption of toxins from the breaking down of the nerve tissue. This uveal irritation would account for the passing hypersecretion.

(4) Treatment has practically no influence on the course of the disease, no matter whether it be of the miotic or operative type. If the view as proposed here be accepted, it is not difficult to see why therapeutic measures, aimed at the relief of hypertension, would be of absolutely no avail.

(5) The causes of this disease lie entirely beyond our knowledge; but it is probable that some systemic selective toxin of unknown nature attacks the optic nerve of eyes so predisposed and produces the results herein depicted.

From the foregoing, I believe that I am justified in assuming that we have to deal with a disease *sui generis* that has points in common with both glaucoma simplex and chronic, clinically noninflammatory uveities, but still differs from both sufficiently to place it in a class by itself. Until further intimate details have been revealed by study, we have no right to give this disease a name.

BIBLIOGRAPHY

Bietti: Ann. di ottal., **40,** 573.
Elschnig: Klin. Monatsbl. f. Augenh., 1895, **33,** 149.
Fuchs: Textbook of Ophthalmology, Wiener Ophthal. Gesell., Nov. 16, 1910.
Ichikawa: Graefe's Arch., **87,** 429.
Lauber: Wiener Ophthal. Gesell., November, 1910.
Leber: Ergebnisse d. Anatomie und Entwicylungsgesch., 1894, **4.**
Markbreiter: Archiv. f. Augenh., 1910, **66.**
Orr: Ophth. Rev., **33,** 33.
Schmidt-Rimpler: Graefe's Archiv, **58,** 563.
Schnabel: Ztschr. f. Augenh., 1904-5, **14.** Klin. Monatsbl. f. Augenh., 1908, 317. Archiv f. Augenh., **24,** 273.
Stock: Klin. Monatsbl. f. Augenh., April, 1908. Klin. Monatsbl. f. Augenh., Beilage, 1910, 124.
Sym: Disease and Injuries of the Eye.
Troncoso: Soc. d'Ophtal. de Paris, 1908, January.

DISCUSSION.

DR. GEORGE F. SUKER, Chicago: Personally, I am somewhat familiar with this type of glaucoma clinically, and I think it is difficult to properly recognize it. Two of Dr. Gradle's patients have been low-grade myopic eyes in aged patients in whom there is an unstable neurotic condition associated with disturbed metabolism. The two cases I have seen have had more or less lenticular involvement and metabolic dyscrasia. That would show there is an interference with the eliminative process of the eye, and may be looked on as a low-grade interference with the stability of the metabolic mechanism of that eye. The tension in these eyes is extremely variable, both as to time and degree. The characteristic disk cupping does not appear until very late and then only a moderate amount. These cases are not suitable for operation, but do best with miotics and a close watching of the elimination and the nerve stability in a broad sense. We should watch for these cases, as I believe there are more of them than we suspect, and if we take our low-grade myopes over 50 years of age, we shall find more of them.

DR. ROBERT SCOTT LAMB, Washington, D. C.: I had a case, reported some ten years ago, and made a record of it. The woman was suspected of having a low grade of simple glaucoma, without tension. That was the impression I got. She was from the country, but I made what record I could and asked her to come back in three months. I gave her pilocarpin and mercury friction, and asked the family physician to watch her renal condition and give her some tonic treatment. Being some distance from her physician, she neglected herself and, some five years later, when I saw her, she had disseminated choroiditis. It seems significant.

DR. MARCUS FEINGOLD, New Orleans: In our clinic at the Touro Infirmary we have had under observation for some time a lady of about 60 years who came to us with rather vague subjective symptoms, but who showed the objective symptom of a rather large and irregularly oval pupil for which we could find no explanation. Examination with the binocular loupe and focal illumination produced no explanation. Our first thought was that we were dealing with glaucoma, but to our great surprise we found on palpation the tension not increased, and, if anything, below normal. Nuclear sclerosis of each lens made a clear view of the fundi impossible; but an impression was gained of an excavation in the temporal portion of the disks. Applications of pilocarpin or eserin produced prompt contraction of the pupils, while cocain increased the existing dilatation. On examination with the Schiötz tonometer, the tension was found to be 18 mm Hg.

DR. WRIGHT C. WILLIAMS, Peoria, Ill.: Dr. Gradle has asked me to say something about this paper, more because I know the patient than because I can throw any light on the subject. Perhaps it will be interest-

ing to know that this woman had a rhinorrhea and was operated on on the basis of a Roentgen-ray picture showing some obscuration of the left ethmoid. The rhinorrhea continued six months and disappeared following this operation. This was done about four years ago. She has been refracted several times by me and has had practically normal vision. She came in with some diminution of vision in September, for which I could find no explanation. She required a little stronger sphere to develop the maximum vision; but I could not find a glass that would give her full vision. Her tension, fields, and urine were normal; and counsel with Dr. Gradle has given this explanation of the trouble. I am not quite satisfied that she has a glaucoma. She has never had any increase of tension, and his reading is the highest ever made on her eyes. I have taken the tension each week and never found either eye over 28, and generally it is 25. Massage always reduces it. The fundus in both eyes is practically the same, yet the vision of the right eye is reduced and that of the left eye not reduced; 20/30 was normal vision for her. Dr. Gradle advised eserin, and after three or four weeks she came in complaining that the vision was worse, but this loss was entirely made up by an increase in the strength of the minus lens. This I took to be the effect of the eserin. I am not familiar with the action of eserin on the eye as far as refraction is concerned; but I believe the eserin made some difference in the refraction. It will be interesting to watch this patient and see whether she gets the cupping and whether her refractive error increases.

Dr. W. B. Lancaster, Boston: I differ from some of the speakers. I agree with Dr. Gradle that these cases do have from time to time a rise in tension over what is for them a safe maximum. It is missed either because the tension is not taken often enough under a sufficient variety of conditions, or because it is not realized that a tension as low as 24 or 25 may be too high for some eyes to bear with safety.

If we knew more about the mechanism of glaucoma the matter would probably be clear. It is a safe assumption that there are a number of factors, some acting in one case, others in others. In the type under discussion there is no marked obstruction of the drainage angle of the anterior chamber by the iris; hence miotics do not relieve. Hence, too, perhaps, the tension never gets very high, because there is always some drainage. The peculiarity seems to be that it does not need to be very high to cause the optic nerve to give way. Therefore, while I believe that operation is indicated, it must be an operation which will reduce tension below, say, 15. Iridectomy will not do this; trephining will. If there were time I should like to report such a case.

It would seem to me that Dr. Suker and Dr. Gradle are too extreme when they discountenance operation. I agree with those who, like Colonel Elliot, think it is the only hope these desperate cases have.

Dr. Wendel Reber, Philadelphia: This gives rise to a lot of interesting questions. Was there an increase in the tonicity of this woman's lens? Has she not some fine choroidal disease? I think likely she has. I believe these cases represent, as Dr. Risley has contended for thirty years, a fine grade choroidal disease. I believe there is some toxemia in this case. If she belonged to my family I should not like anyone to operate. I confess a myotic does not do much good, but she has the other type that may easily flare up at any time. They do not all occur in old people. Dr. Francis and I have studied a case in a man of 35. While there was an excavation, he has never had an increase in tension, but if any of you would look at him you would recognize the saucering, if not the cupping. I believe he will bear watching, but I don't think it

wise to operate. I don't believe 23 to 24 mm. of mercury in a man of 60 is enough indication for operation. It does not necessarily follow that this is an abnormal tension. Eighteen might be an indication in some eyes and thirty might not be in others.

Dr. Edward Jackson, Denver: There is one thought I would like to inject into the discussion, and that is that the tension that is normal is not a fixed tension, although it may vary within narrow limits. The thing which is normal is a certain balance of intra-ocular tension, and other forces influencing the nutrition of the eyeball, as the blood pressure. This balance may be overthrown if the tension rises too much, and in other eyes it might be overturned if it did not rise at all, but the opposing factors failed. A good many years ago, I saw a case of this sort which impressed me strongly, that an eye could have a typical and marked glaucoma cup, without any rise of tension at all. There was absolute blindness in this case, of a man over 70, and so far as we could observe there was no evidence of rise of tension. A case that would come nearest to this type I have seen in the last two years, since using the tonometer. He has never had a rise of intra-ocular tension, which generally runs from 20 to 24 mm. That eye has a perfectly typical glaucoma cup, and greatly narrowed field, and the process has seemed to be held in check by pylocarpin, and when he stopped using it several weeks, the eye got absolutely worse. That patient had an unusually low blood pressure. I do not think his systolic pressure has been up to 120 at any time, although the man is about 90 years of age, and it has been below 110. I would not want to draw a line between this class of cases and the class of glaucoma. I want to mention with respect to this case that his other eye has shown a distinct rise of tension, and his other eye was blind with a glaucoma cup when he first came to me.

Dr. Hiram Woods, Baltimore: I want to give briefly an account of two cases watched for a number of years. One was that of a woman of 60 years of age, who, during three years, had some half dozen or more additions made to a myopic glass by an optometrist. She had a myopia of about 6 diopters, rather large, strictly regular myopic crescents with a typical pale chronic glaumatous cup. Vision was reduced to about one-half. I watched her seven or eight years. I saw her at every hour in the day, except from 11 o'clock at night to 8 in the morning, hoping to find some time when the tension might be increased. I never detected the slightest elevation in tension. She had some mysterious abdominal trouble (I could not find out what), and she went along that way for eight or nine years, when her field began to contract. The last time I saw her it was down to 20 degrees, without change in the optic nerve. The essential point in the case is the long retention of a certain amount of vision, although the ophthalmoscope showed typical glaucoma and then, without apparent reason, rapid contraction of fields to almost complete loss of sight.

The other case was an old lady who had lost her left eye in early life from central choroidoretinal atrophy. She had been under the care of Drs. Derby, Knapp, Chisolm, Webster and others. She had a diagnosis of glaucoma from one or two, while the others flatly contradicted it. Her refraction was hyperopic astigmatism, field was normal. When the tonometer came into existence she would not allow me to use it. Here is what happened. After she had gone along eight or nine years without contraction of field or elevation of tension, her eye suddenly developed high tension with a large and rapid contraction, When the field contracted and tension became elevated, I sent the notes to Dr. Bull and he advised against operation, basing his decision on: (1) The fact that the field at

one point was contracted to within 10 degrees of the center; (2) the presence of certain associated abdominal symptoms which were suspected of pointing to malignancy, and (3) she was a chronic gouty subject. Field contraction progressed until sight was blotted out. At no time was she willing to submit to operation.

Here are two cases, one myopic, the other, hyperopic. Both had ophthalmoscopic glaucoma, yet both preserved the same degree of visual acuity and retained stationary fields for long periods. Then both suddenly developed glucomatous symptoms. Were they glaucoma from the beginning? I confess I do not know.

Dr. Gradle (closing discussion): I think I am in the wrong school. Everybody has been trying to connect tension with the disease. It has nothing to do with it. I don't believe it is the essential factor. In the cases I have seen, the tension has been easily reduced by massage. I do not believe we have the cupping as the result of pressure at all, but that the pressure is secondary and that the disease is in the optic nerve, and the tension is a secondary factor induced possibly by the choroiditis, as Dr. Reber suggested. It disappears in a short time and does not play an active rôle in the disease. The picture is entirely different from glaucoma, and is an optic nerve disease, and the rise in tension has nothing to do with the disease. I reported it as a case of glaucoma simplex without perceptible rise in tension, merely because that was the name in common usage.

CONGENITAL PARTIAL DEFECT OF RETINAL PIGMENT LAYER OF IRIS IN BOTH EYES

M. Feingold, M.D.
NEW ORLEANS, LA.

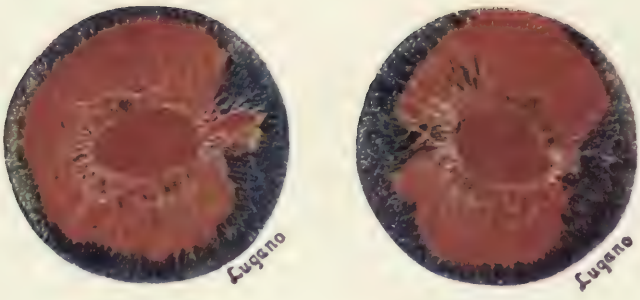
Two methods of examination that allow the study of the iris in the living eye have, during the past few years, very greatly contributed to our knowledge of the anatomy of this organ. Following the fundamental researches of Fuchs regarding the normal anatomy of the iris, increased attention was devoted to the study of the anterior surface of the iris and its details by means of the binocular loupe of Zeiss and the corneal microscope—investigations that have brought forth some very interesting facts. On the other hand, a method already suggested by von Graefe was facilitated by the rapid advances in electric engineering, which, in producing Sachs' lamp, Hertzell's diaphanoscope, Würdemann's transilluminator, von Pflugk's and Verhoeff's instruments, made transillumination so easy of execution.

Transillumination, as now practiced, consists in allowing more or less strong light to enter the eye through the intact sclera and to examine the interior of the eye and with it the iris under these new conditions. Interesting and important facts have already been established by this principle of examination and Vuellers' paper marks a new epoch in the examination of the iris. He could show that the iris, in consequence of disease, would often lose its power to absorb light in such manner that transillumination would show up as translucent spots the places where the iris had been affected by traumatism or disease. But it is not with these results of transillumination that we are concerned at this moment, interesting though they are. In this same paper Vuellers speaks of "patients that complain of symptoms of dazzling, especially in strong sun light." In the eyes of such patients transillumination is said to show "absence" of retinal pigment. Little more is said about these cases, except that an excellent picture is furnished of the condition on Plate II, Figure 7, accompanying his article. Nohting further of any note appeared in the literature concerning the iris and transillumination, until Ruebel published his extensive and

painstaking researches. One of the conclusions he arrived at is: Permeability of the iris to light depends not so much on the condition of the pigment epithelium as on the condition of the iris stroma. Commenting on this view, Fuchs elaborates on the many elements that go to make up the greater or lesser permeability of the iris and arrives at the conclusion that with "equal pigmentation and thickness of the iris stroma the transparency of the iris depends on the retinal layers and the real good transparency always depends on defective pigmentation of this layer."

The following case, although observed only clinically, proves, I believe, that, all things being equal, the condition of the retinal pigment determines the ability of the iris to allow light to pass through or to absorb it.

Mr. H. O. W., aged 29 years, called September, 1911, with the following history: In 1894 he had sore eyes — "just ordinary sore eyes"—there having been an epidemic at the school at that time. Since then he thinks eyes have been sensitive to light. In 1901 his eyes were inflamed and an ophthalmologist ordered glasses for constant use, but he used the glasses only for near work. In 1905 the glasses were changed, and again in 1910. No other illness or defects were manifested. Parents are not blood relatives. V. R. E. 5/9? V. L. E. 5/6? Retinoscopy under hematropin: R. E. $+2.50+\bigcirc 1.00$ Axis 105 degrees. L. E. $+3.00+\bigcirc 1.00$ Axis 75 degrees. Subjective: R. E. $+2.50\bigcirc +1.0$ Axis 120 degrees 5/5. L. E. $+2.50\bigcirc +1.0$ Axis 60 degrees 5/5. Iris was gray-blue. Hair was black. Pupils were irregularly oval with the long diameter approximately at axis 15 degrees in the right eye and 165 degrees in the left eye. Pupils reacted apparently along the whole pupillary margin and when viewed with the binocular loupe the movement seemed more marked in the left eye. There being no posterior synechiae, homatropin brought about prompt dilatation and the dilated pupils retain relatively the original form, but the dilatation seems to be most marked at the points of elongation of the pupil. In the right eye the greatest effect is on the nasal side; in the left eye the greatest effect is in the lower temporal quadrant. With the binocular loupe the pupillary margin of the iris is normally thick only in two places in each eye, these places corresponding to the ends of the longest diameter of the oval pupil. In the other places the iris is thin, the meshwork of fibers being less dense and the single fibers thinner. On the anterior surface of the iris the pupillary zone is separated from the ciliary portion by an irregular condensation and interlacing of the trabeculae that are on the whole rather fine and appear more whitish than the balance of the iris. Only here and there are the fibers more thick and heavy. In several places distinct crypts are noticed in the ciliary portion of the iris.


Lugano
Lugano

Under ophthalmoscopic examination each eye showed a normal, healthy, uniformly light fundus. But the light thrown into the eye by the ophthalmoscope produced a most striking and strange effect—a spectacle at first rather puzzling.[1] Instead of the expected uniform red fundus reflex showing up in the more or less oval pupil, a much larger, irregularly shaped red reflex of different intensity in different places presents itself. Closer analysis of this phenomenon by means of the Sachs lamp or the Würdemann transilluminator proved it to be due to the fact that light is leaving the fundus not only through the pupil as in the normal eye, but in this case also through other parts of the iris, and through these parts in varying intensity depending on the structural condition of the different spots.

The peculiar irregular outline described is due to the fact that in each eye a part of the iris near the ciliary border is entirely impervious to light. In the balance of the iris small areas here and there are more or less opaque similar to those in the peripheral portion. These areas are especially noticeable as small, reddish-gray spots in the sphincter region of each iris. On the other hand, areas can be made out in the ciliary part of the iris that are much brighter than the surrounding iris and show up almost as red as the pupil, areas which undoubtedly correspond to the crypts.

White it is by no means an unusual occurrence to find, in eyes that show evidence of disease or traumatism, more or less circumscribed or limited[2] defects in the iris, which allow light to pass through, the condition underlying the above described is apparently of an entirely different nature. There is no history of any injury, or of any eye trouble, for the "inflammation" mentioned in the history was apparently of a very mild nature belonging to the acute conjunctivitis class. The second inflammation mention seems to have been of even less importance, since the ophthalmologist ordered only the correction for the ametropia. The eyes in addition are free from any defects attributable to inflammation. In these instances in which the iris becomes permeable to light in consequence of inflammation, such as iritis, these spots are usually marked by the presence of posterior synechiae.

The condition is bilateral and is almost symmetrical in the two eyes. Defects of the iris leading to greater permeability to light following illness or tramatism are hardly ever bilateral and still less symmetrical. There seems to be a certain relation-

1. The accompanying illustration attempts to reproduce the impression gained when the iris was viewed with the electric ophthalmoscope and +15 to +20 D.
2. An exception to this forms the unique case of Stölting, in which uniform defect of retinal epithelium of the iris developed after malaria.

ship between the direction of the long axis of the oval pupil and the direction of the astigmatism—a relationship which is the same in the two eyes. Astigmatism, though sometimes acquired, is most of the time congenital and nearly always so when symmetrical in the two eyes.

From all this the conclusion seems to be inevitable that the condition described is not due to traumatism or disease, but due to some congenital factor which produced the almost identical and symmetrical defects in the iris of the two eyes and also produced the symmetrical astigmatism which holds a certain fixed relation of its axis to the long diameter of the oval pupil.

Congenital defects in the iris are by no means rare, but those defects of the iris that allow light to pass through are generally of an entirely different nature. Fuchs classified these eyes into albinotic and semi-albinotic eyes and Vuellers' lines quoted above seem to refer to a similar condition as shown by the picture on Plate II, Figure 7, of his article. In all these cases of partial or complete albinism the defect is one comprising the whole retinal pigment layer, and for that reason the defect in the iris is uniform, but of varying degree in different cases, as shown by the more or less intense but uniform permeability to light on transillumination. In the case here reported, transillumination proves that the defect is by no means uniform. In the peripheral portion of each iris there is a normal area which is almost entirely impervious to light. The balance of the iris allows light to pass through almost unhindered, excepting in those smaller but apparently not quite normal areas in the sphincter portion which show up as reddish-gray areas on transillumination.

Focal illumination and the binocular loupe fail to produce an explanation for the peculiarity of the iris in this case. The structural characteristics of the anterior layers of the iris (stroma) are not in any way different from those of any other iris, and certainly do not so abruptly change from place to place as to explain why sharply outline areas in which the iris is entirely impervious to light should adjoin portions that offer almost no obstacle to transillumination.

The cause of the peculiar appearance of the iris on transillumination must therefore be due entirely to some form of defect of the retinal pigment layer on the posterior surface of the iris. This way one can understand that the pigment epithelium is normal, or almost so, in those areas at the periphery in which the

iris shows up dark on transillumination; while the portions that allow light to pass through apparently have pigment epithelium which is more or less defective in different parts. It must be understood, however, that the word "defect" as used above is not to mean entire absence of the pigment epithelium cells, but to signify that the cells are not entirely normal in all their characteristics.

The irregularity and oval shape of the pupil is apparently also independent of the condition of the stroma of the iris; because close inspection with the loupe reveals no sufficient cause for these changes. However, these changes can be easily explained if we remember that both sphincter and dilating muscles of the pupil are embryologically derived from the pigment epithelium. In this case one can imagine that the uneven defect of the retinal layers of the iris produced not only differences in the transparency, but differences also in the tonus and condition of the muscles in the different sectors of the iris and thereby the irregularly oval pupil.

This case, apparently the only one of its kind in the literature at my disposal, would then prove the contention of Fuchs that the retinal pigment layers play the principal rôle in preventing light from passing through the iris. It shows on the other hand that even a blue iris, one poor in pigment, forms some obstacle to light, varying in degrees according to the thickness or condition of the stroma.

BIBLIOGRAPHY

Fuchs, E.: Beiträge zur normalen Anatomie d. menschl. Iris, Arch. f. Ophth., 1885, **31**, Part 3, 39.

Stölting: Folgen einer Chininvergiftung am Auge, Arch. f. Ophth., 1903, **55**, 85.

Vuellers, H.: Neue, diagnostisch wichtige Resultate bei Durchleuchtung des Auges mit der Sachsschen Durchleuchtungslampe, Ztschr. f. Augenh., 1907, **18**, 215.

Ruebel, E.: Ueber die Durchlässigkeit der Iris für Licht bei der diaskleralen Durchleuchtung im normalen und kataraktösen Auge, Arch. f. Ophth., 1912, **82**, 317.

Fuchs, E.: Normal pigmentierte und albinotische Iris, Arch. f. Ophth., 1913, **84**, 521.

DISCUSSION

Dr. H. Gradle, Chicago: Dr. Feingold dealt with the congenital absence of the retinal pigmentation of the iris. I have recently observed a case in which the reverse occurred. A man with a brown iris developed an iritis, following which the iris became almost entirely blue. This persisted for some time. Later the pigment began to advance toward the pupil from the iris root. The pupillary margin of the iris, and in spots scattered around there, is still blue, but there is a steady and uniform advance of the brown pigmentation from the periphery.

Dr. Feingold (closing discussion). I should like to emphasize the points that compelled me to assume that the condition found in this patient was due to some congenital cause. The defect in the pigment of the posterior layers of the iris apparently has a causative relationship

to the shape of the pupil. It is this defect which makes the pupil of each eye appear irregularly oval. Since, on the other hand, the direction of the astigmatism holds in each eye a fixed relation to the longer axis of the oval pupil, and since the low amount of the symmetric astigmatism could only be congenital, it is only natural to assume that also the pigment changes are due to nothing but a congenital cause. In using the word congenital it is not meant that the condition actually existed at the time of birth; but it also may be that this individual was born with some congenital condition predisposing and inducing this peculiar change in the retinal pigmentation of the iris.

SCLERAL TREPHINING IN DETACHMENT OF THE RETINA

JOHN GREEN, JR., M.D.
ST. LOUIS, MO.

The treatment of detachment of the retina, whether medical or surgical, has always proved extremely unsatisfactory. There has seldom been a more striking example of unanimity of medical opinion than the replies to a questionnaire of Vail (1913) relative to the curability (or incurability) of this disorder. With only an occasional dissenting voice, nearly 300 American ophthalmologists asserted in the strongest terms their belief in the entire inefficacy of any form of treatment. This collective view tends to confirm convictions which most of us, I imagine, have long held, viz: that detachment of the retina is, with rare exceptions, an incurable disease.

In this paper no attempt will be made to discuss the various theories of causation. I shall omit also a description of all of the medical and most of the surgical methods, which, from time to time, have been proposed for its relief. My purpose is to review the application of scleral trephining to detachment of the retina, and to comment on another operation, "pre-equatorial sclerectomy," which is, in effect, a modified scleral trephining.

It is not without interest in this connection, that the operative method most in favor with American ophthalmologists (as determined by Vail's investigation) is simple puncture of the sclera, the knife penetrating to the subretinal space in order to afford an opening for the immediate egress of the subretinal fluid. After the knife is withdrawn, the lips of the wound come into close contact, so that prompt agglutination soon puts an end to drainage.

The possibility of effecting a permanent channel, by means of a trephine opening in the sclera for drainage of subretinal fluid into the subconjunctival space, seems to have occurred almost simultaneously to a number of operators.

Schreiber[1] appears to have been the first to propose this operation. After experimentation on rabbits, he concluded that it was possible to establish a permanent passage for the flow

1. Schreiber: Klin. Monatsbl. f. Augenh., **52**, Bd. 1, 120.

of subretinal fluid into the subconjunctival space. He inserted a strong traction suture in the sclera at a point corresponding to the insertion of the inferior rectus. The globe was then rotated upward and a triangular conjunctival flap was formed below the suture. The sclera was denuded either to the right or left of the inferior rectus and trephined, followed immediately by puncture of the choroid. A conjunctival stitch was them inserted to draw together the edges of the flap. Apparently without knowledge of Schreiber's work, Tiffany[2] on Jan. 15, 1914, successfully performed scleral trephining in a patient with traumatic detachment. A small Elliot trephine was used to remove a scleral button over the center of the detachment. On removal of the button, the protruding knuckle of choroid was clipped away, followed by escape of serous fluid and a little vitreous. Tiffany contended that the retina was forced into the opening of the choroid, "coming into contact with both choroid and sclera. At this point it forms a firm and permanent adhesion."

W. R. Parker[3] reported a detachment of the retina occurring with neuroretinitis. For a year vision had been growing worse and was at the time of the operation 4/60. Parker laid back a conjunctival flap, trephined the sclera, punctured the retina and choroid with a sharp knife, allowing serous and glairy fluid to escape. Eventual result V. = 6/12; field partially restored.

In a subsequent communication, Parker[4] recounted his experiences in fifteen operations on eleven patients, the first operation being performed May 19, 1913. A triangular flap of conjunctiva is made at a site corresponding as nearly as possible to the lowest portion of the detached retina. Before releasing the flap from the grasp of the forceps, a suture is inserted, both for purposes of traction and eventually to replace the flap. A 2 mm. trephine opening is made, and after removal of the button the choroid, or choroid and retina, is incised. The free escape of the subretinal fluid may be followed by a few drops of vitreous. After replacement of the flap, using supplementary sutures if necessary, the patient is returned to bed and kept recumbent for ten days. In summarizing results, Parker found that vision was improved in four, unimproved in five, and made worse in three cases. In eight cases the visual fields were improved; in three they were made worse. In three cases very

2. Tiffany: Ophth. Rec., **23,** 132.
3. Parker: Arch. Ophth., **43,** 489.
4. Parker: Trans. Section on Ophthalmology, A. M. A., 1915, p. 106.

good results were obtained. Parker states that his object "has been to secure an opening in the sclera and choroid which would allow free drainage of the subretinal fluid for a sufficient length of time to permit the retina to become re-attached. If the secretory process is dormant and all the subretinal fluid evacuated at the time of the operation, re-attachment may occur without edema of the conjunctiva. But if the secretory process is active, or if any amount of the subretinal fluid is left behind, a certain amount of reaction must necessarily follow or the retina will again be separated from the choroid and failure result. In each of the cases in which a good result was obtained, the operation was followed by a marked edema which did not entirely disappear until the tenth day." Parker notes that the operation seems to be without danger to the integrity of the globe and that "the best results were obtained when escape of the subretinal fluid was followed by a discharge of a small amount of vitreous."

A modification of this method was proposed by Curtin,[5] which he first tried on a patient with a spontaneous detachment of recent occurrence. After ten days of futile treatment (rest in bed, 0.5 per cent. atropin daily, pressure bandage, pilocarpin sweats), Curtin trephined the sclera, but *without puncturing the choroid or retina.* The conjunctival flap was replaced with two sutures and the patient returned to bed and kept recumbent for ten days, the eyes being closed with a pressure bandage. At the end of this time the ophthalmoscope showed no evidence of re-attachment. Twenty-five minims of fluid was then aspirated, by introducing the tip of a hypodermic syringe into the subretinal space through the scleral opening. Immediately the retina was found re-applied and prompt recovery ensued, with final vision 20/30. During convalescence the conjunctiva over the scleral opening was observed to be slightly chemotic, evidence of continuous drainage of the subretinal fluid into the subconjunctival space. At each dressing the conjunctiva over the opening was massaged to keep the opening patent and, as Curtin expresses it, "to facilitate the drainage between the subretinal and choroidal space and the subconjunctival tissues." Curtin claims the following advantages over simple trephining:

(1) Small amount of traumatism, the operation being done in two stages.

(2) No injury to the retina and no loss of vitreous.

---

5. Curtin: Ophth. Rec., **24**, 68.

(3) Absence of marked reaction.

(4) The possibility of the examination of the fundus during and immediately after operation.

(5) The possibility of repeated aspirations should the subretinal fluid re-accumulate.

In an elaborate paper in which various views of detachment of the retina are described and discussed, Thomson and Curtin[6] find that "the evidence on all sides . . . is in favor of some operative procedure which withdraws the subretinal and suprachoroidal fluid." Thomson, in a subsequent paper,[7] sums up their views as follows:

"The idea has been to get rid of the supra-choroidal fluid primarily, and, secondarily, to forcibly aspirate the subretinal fluid as completely as possible, so as to allow the retina to reassume its normal position. The filtration through the trephine opening may then prevent more fluid from accumulating, and forcibly separating the retina from its position secondarily."

Three cases are reported in detail, of which two were successes. The operation was also done in ten other cases, all of which were old cases of detachment, and the patients were operated on to determine whether the operation had any deleterious effect on the integrity of the eye.

Ohm,[8] Brieder,[9] and Lewis[10] report single successful results following simple trephining.

An operation similar in principle to trephining has been devised by Holth and called "pre-equatorial sclerectomy."[11] It is thus described: A meridional incision is made in the sclera with a convex scalpel, or the point of a lance knife. Into this slit the blade of a Vacher punch is inserted and an opening of any desired size is made in the sclera. He reports good results in myopia and myopia with retinal detachment.

Schiötz[12] reports twenty-one cases of pre-equatorial sclerectomy in retinal detachment. The conjunctival flap is cut out and down. After incising the sclera meridionally, a Holth trephine punch is used to excise the sclera down to the choroid (in some cases the choroid is also incised). His results were as follows: Five cases improved, nine cases continued as before operation, six cases were made worse. The final result showed

6. Thomson and Curtin: Trans. Section on Ophthalmology, A. M. A., 1915, p. 87.
7. Thomson: Ann. Ophth., 1916, January, 20.
8. Ohm: Ztschr. f. Augenh., **33**, 288.
9. Brieder: Arch. Ophth., **44**, 552.
10. Lewis: Jour. Ophth. and Oto-Laryngol., 1916, February, 52.
11. Holth: Trans. of the 39th Ophthalmological Congress, Heidelberg, 1911.
12. Schiötz: Norsk. Mag. f. Lægevidensk., **76**, 1225.

three cases improved, five in the condition they were before operation, eleven made worse.

Almost every year the literature contains reports of spontaneous recoveries from detachment of the retina. This fact should be borne in mind in crediting to any method of treatment a successful issue.

CASE REPORTS

CASE 1.—Miss L. K., aged 55, sewing machine operator for a bag factory, consulted me June 28, 1916. Aside from moderate myopia, which dated from early youth, she knew of no ocular disability. She wears in both eyes a —5.5 sphere. About May 15, 1916, she began to observe "spiderwebs" before her left eye. A week later the webs disappeared, but soon returned. R. E. —5.5 sph. V.=6/8. L. E. —5.5 sph., V.—hand motion at 60 cm. The ophthalmoscopic examination showed in the right eye a few dust-like opacities in the anterior part of the vitreous. Moderate conus, out. The left eye presented extensive detachment of the retina. Red reflex was obtained only up and temporally. Field (taken with pocket flashlight) was greatly contracted. The patient entered the hospital and was operated on June 29, 1916. A conjunctival and tenonian flap was dissected up between the insertion of the inferior and internal rectus. On removal of the 2 mm. scleral button the choroid presented. A very delicate pair of straight forceps was passed, closed, into the subretinal space. On allowing the blades to separate, about 30 minims of yellowish fluid, slightly glairy, flowed out. The flap was replaced and sutured in position with two sutures. The patient was kept very quietly in bed, though no special effort was made to keep her in any one position. At the end of the first week she could count fingers at six feet. The end of the second week, however, witnessed a return of the cobwebs and vision was no better than before operation. The patient declined to remain longer in the hospital, and the result would have been classed as an unqualified failure, if the occurrence of a detachment in the other eye, July 20, had not brought her again to observation. Opportunity was thus afforded to observe the eye first operated on, the vision of which had slightly improved, and on October 30 was 1/50. It was found also that it was no longer necessary to employ a flashlight to outline the field, as the patient readily recognized the ordinary 10 mm. test object. Ophthalmoscopically, the vitreous was found filled with very numerous gray particles and the retina was still detached downward and outward.

CASE 2.—Mrs. F. L., aged 60, consulted me April 15, 1916, complaining of failure of vision in the right eye beginning four months previously. A nearly central corneal scar prevented good vision in the left. Recently vision of the right had sud-

denly become worse, R. V.=6/50. L. V.=1/25. The right upper field was lost. The right vitreous was full of a fine network of opacities. There was a flat detachment of the retina in its lower part encroaching upon, but not including, the macula. Rest in bed for two weeks, with pressure bandage and potassium iodid internally, proved an utter failure. On May 2 the sclera was trephined between the insertions of the external and inferior recti, followed by the escape of a few drops of fluid. Following the Thomson-Curtin technic, the flap was replaced without perforating the choroid.

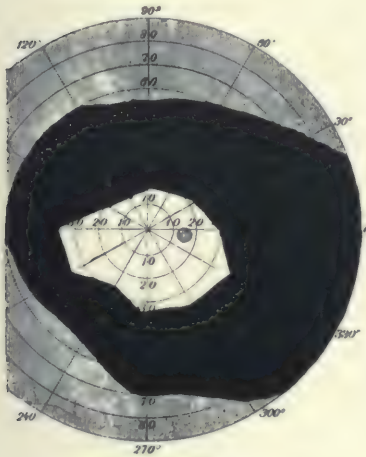
Two weeks later (the patient meanwhile having been kept in bed) vision had risen (with +1 sph. +0.5 cx. 180) to 6/20, and a little tongue of vision had projected itself into the upper blind field. At the second operation (May 17) I was unable to locate the trephine opening through the thickened flap and hence was compelled to dissect up the conjunctiva. A small platinum hypodermic needle was introduced into the subretinal space, withdrawing ten or more drops of a slightly glairy fluid. The flap was replaced with three sutures and the patient returned to bed. Vision remained at 6/20 for two and one half months with nearly complete restoration of field. Ophthalmoscope showed that the retina had re-attached except at one small area below. The patient was allowed to return to her home in the country, but came back September 23 with a secondary detachment and vision fallen again to 6/50. There was a complete loss of the upper field.

Case 3.—A. O., a male, aged 49, was first seen Aug. 25, 1915. He stated that he had received a blow on the right eye five months before. R. V.=6/16. L. V.=6/15. Right eye showed a retinal detachment to the outer side and below. There were numerous fine floating opacities in the vitreous and some fixed thread-like bands extending transversely. Patient was treated with subconjunctival injections of sodium chlorid 1 per cent. and cyanid of mercury 1-4000 without improvement. Oct. 4, 1916, scleral trephining downward and outward with perforation of the choroid was followed by a gush of watery fluid. Healing was prompt and there was no undue reaction. During the course of the medical treatment vision had fallen to fingers at 1 m. After the trephining it rose to 1/12. There was no material change in the visual fields.

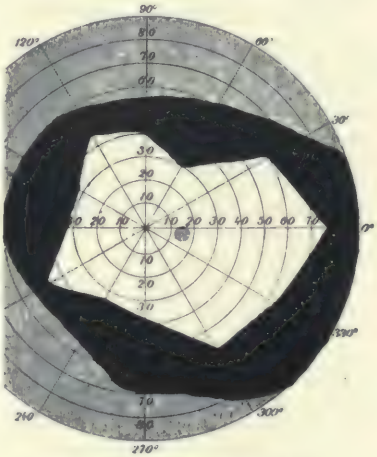
Case 4.—This case occurred in the service of my colleague, Dr. William F. Hardy. The patient, J. M., a female music teacher, aged 67, came to the outpatient department of the Washington University Dispensary Sept. 12, 1916. She complained that for the past month objects seen with the right eye seemed "green and feathery." Examination showed a detachment of the retina in the lower inner and outer quadrants. Transillumination was negative. Vision +3 sph.=20/75. Patient was admitted to the surgical ward September 28 and was operated on by Dr. Hardy, October 3. Following the

removal of the trephine button, delicate forceps were passed into the subretinal space followed by an escape of thirty minims of glairy fluid. There was very slight reaction, and the patient left the hospital six days after operation. The last visual test was November 18: R. E. +3.75 sph. V.=20/60. There was no increase in the field. Ophthalmoscopically there is no change whatever in the position or extent of the detachment.

Case 5.—This case concerns the right eye of Miss L. K., whose left eye forms the subject of my first case report. On July 20, about ten days after leaving the hospital, following operation on the left eye, she suddenly went blind in the right. The next day she was led into the clinic. The retina was very extensively detached, in fact, it was impossible to outline the disk, and no red reflex was obtainable. Vision = hand motion at 1 m. In consultation with Dr. A. E. Ewing, it was determined to trephine. The operation was performed without inci-



Case 5.—Field before operation.



Case 5.—Field after operation.

dent. A 2 mm. trephine button was cut out between the insertions of the inferior and external rectus muscles. A fine pair of forceps was introduced, closed, into the subretinal space and the blades allowed to open slightly. There was an immediate gush of about ten drops of a glairy fluid which looked like fluid vitreous. The conjunctival flap was replaced and the patient returned to bed, where she remained quietly supine for two weeks. There was no improvement at the time she left the hospital. The folds of the retina were still visible in all parts of the field. The patient was put on syrup of hydriodic acid and carefully watched. About September 15, there was a good red reflex from all but a small portion of the lower part of the fundus, and vision had risen to 6/50 with improvement in the field. Improvement continued and on Dec. 8, 1916, Vision = 6/10 with —5.5, and the field was nearly full, as shown by

appended diagram. Ophthalmoscope showed a reattachment complete except for a small area in the lower part of the fundus.

SUMMARY

Cases 1 to 4 should be classified as failures, there being no permanent reposition of the retina and no material improvement in either vision or field.

Case 5 represents to date a success, but a success that at any time may be converted into a failure.

Cases 1 to 4 were all old detachments, associated with evidences of secondary inflammatory and degenerative changes.

Case 5 was operated on within three days of the occurrence of the detachment, the patient having been previously operated on for old detachment of the fellow eye (Case 1).

If we adopt an operation which withdraws the subretinal and suprachoroidal fluid and, as Thomson and Curtin have remarked, the evidence is all in favor of some such procedure, is it not more rational to operate as soon as possible after the occurence of the detachment, rather than to temporize with nonsurgical methods, thus letting pass what may be the most favorable time for operation?

DISCUSSION

Dr. William H. Wilder, Chicago:—It seems to me that one of the most significant sentences in this excellent paper of the essayist is the one that states that "almost every year the literature contains reports of spontaneous recovery of detachment of the retina." Many of us can testify to the truth of this, and it should be borne in mind in accrediting to any treatment a successful issue.

As I understand it, the discussion is to be limited to this particular form of treatment of detached retina, namely, scleral trephining.

My experience in this has been limited to about six cases and I am free to say is not very encouraging. One of my cases was witnessed by the essayist some time ago in Chicago. The patient, a middle-aged, healthy woman, had spontaneous detachment of the retina of the right eye that was not improved by the usual treatment of several weeks' rest in bed, with sweats, diuretics, etc. She was fully informed of the seriousness of the condition and consented to operation. Scleral trephining was done at a point beneath the external rectus muscle, after drawing the tendon aside, and the choroid was penetrated. Healing was uneventful, and the condition of the eye was much improved temporarily, but only for a short time. The detachment recurred after a few weeks, and although she had no acute symptoms, there developed within a month or two a peculiar insidious uveitis which resulted in binding the iris firmly to the lens, so that it was impossible to dilate the pupil. I have seen a similar condition following trephining for glaucoma and am at a loss for an explanation.

Some of my cases have shown temporarily brilliant results, but the ultimate outcome was disappointing. One patient consulted me because of a detachment that had occurred spontaneously or after a rather slight

iar, two weeks before. When first seen, the vision of the affected eye was greatly reduced, because of an extensive detachment of the retina below and also because of a very hazy vitreous. After two weeks of the usual treatment in bed with sweats, etc., he was able to count fingers a few feet away, and the extent of the detachment could be determined somewhat by the ophthalmoscope.

He consented to an operation after being informed of the uncertainty of it and of the slim chance of recovery without it. Scleral trephining was done by making the opening beneath the inferior rectus tendon which was drawn aside and the choroid coat was perforated after removing the button of sclera. Recovery from the operation was prompt, and in a few days the eye began to clear rapidly. A beautiful result followed. The retina replaced itself so perfectly that it was impossible to determine with the ophthalmoscope that there had been any detachment. The patient was kept in bed for two weeks after the operation and then allowed to go about. Central and peripheral vision returned to nearly normal.

About two weeks later, while in New York, the detachment recurred. Dr. John Shannon, after learning of the result I had obtained, repeated the scleral trephining with a similiar happy result, but this, too, was only of temporary duration, for in a short time the retina again became detached.

Another case that illustrates so well the significant sentence in the author's paper to which I have alluded was seen within the last two years.

An elderly lady, with detachment of the retina in the lower and temporal part of the left eye, consented to operation rather than to rest treatment of weeks in bed. After a few days in bed, she noticed her vision improved and the operation was postponed. Within two weeks the retina had replaced itself entirely, and she left the hospital with nearly normal vision and the fields much improved. This fortunate result, which might have been attributed to an operation, if it had been done, lasted about two months, and then the retina again became detached, although the patient still retains a fair degree of central vision that may at any time be lost.

It seems to me that in these cases we must tell the patient frankly the gravity of the condition and explain the great uncertainty of the result from any treatment whatsoever, and then let him decide what he will have done. None of us on this side of the water seems to have achieved the remarkable results of our German confrères, notably those of Deutschmann.

Dr. Jones, Cumberland, Md.:—Over three years ago a man of about 50 presented himself to me with a detachment of the retina so far advanced that it looked like a cataract. He was entirely blind. I had just recently had the experience of failing with three beautiful cases of detached retina, and I had been thinking over the matter, and I told him if he was willing to submit to something which, to my knowledge, had never been done, I thought that he might see. Like a sensible man he asked: "Can it make me worse?" I said, "No." He said, "Go ahead and shoot." I had an idea to produce a continuous subjunctival drainage, and the method I adopted was under cocain and adrenalin, to grasp the conjunctiva close to the cornea and draw it forward, and with an electrocautery point I burned through. There was an escape of twelve or fifteen drops of yellowish fluid. And then, when the grasp was released the hole in the conjunctiva slid out further than the hole through the sclera, and we had a subconjunctival hole practically the same as the trephine. The idea I had was twofold. First, to make a hole that would not close quickly, and second, to set up an inflammatory reaction to aid in the reattachment. I learned later that after trephining there have been cases in which the

result was spoiled by a hemorrhage, so I thought burning would arrest the hemorrhage. Then, as an extra precaution, I made an injection of cyanid of mercury, which prevents infection and which elevates the nutritional tone of the eye. By itself the conjunctival injection does no good. When I opened up the lid I found he could see. He was allowed to go home and to do as he pleased, except he was to avoid exercise. In the British Ophthalmological Society two years later, this method was brought out jointly by Maitland Ramsay and Leslie Patton, and many of those men reported cases cured with cautery and subconjunctival injections. My patient still has good vision.

Dr. George F. Suker, Chicago:—I just wish to report an experiment in a case of double detachment of the retina. I have a case in hand which presents a bilateral detachment in the lower periphery of both eyes. We know the subretinal fluid contains albumin. By means of a hypodermic syringe, so as not to do any more damage than was necessary, I abstracted the entire aqueous which is nearly three-fourths of a syringe; so much, in fact, that the eyeball collapsed. I repeated that once a week, and whereas the first abstraction in that one eye was highly albuminous, the last was negative, as far as the albumin content was concerned. When the eyeball regained its normal tension, a trephining was done over the sight of the detachment. We were unable to get any fluid with a syringe, the retina was not any further detached, nor was it changed, but evidently had formed a sort of adhesive band and the case is not worse than it was before. We followed the same course in the other eye, and I have fully as good a result. To be sure, this was an experiment.

Dr. G. T. von Colditz, Chicago:—I would like to call your attention to an interesting case which came under my observation while assistant in the eye clinic at Heidelberg about twelve years ago. The patient, a male, aged about 50, was a mason by trade. He had a double detachment of the retina when admitted to the hospital the first time, with only very small amount of vision in either eye. Patient was put to bed and received all the usual treatments except surgical intervention. After three months of unsuccessful treatment, the patient was discharged as incurable. He was told to keep as quiet as possible and not to go back to his former occupation, as he might otherwise lose all his vision. Two years later the patient returned to the hospital for reexamination, telling us he had been working hard at his usual trade. We found a complete reattachment of the retina and good vision.

Dr. R. S. Lamb, Washington, D. C.: I have only one small fact to contribute. It is my honest belief the reason some of these reattachments do not stay for a while is that the intra-ocular tension is not sufficient to hold the retina in place, and it is my suggestion that we attempt to increase the intra-ocular pressure. I believe that can be done in two ways: (1) By increasing the fluid of the body by the feeding of salt, and (2) there is no doubt that thyroid and suprarenal will increase the secretion from the posterior secreting surface of the ciliary body.

Dr. W. B. Lancaster, Boston:—My experience has been limited to less than a dozen cases, but I have had occasion to observe a good many patients operated on by my colleagues at the Eye and Ear Infirmary at Boston, and briefly the conclusions that so far have been impressed upon me are that we can in a very large percentage of cases produce an immediate reattachment by some method of scleral puncture and drainage provided the drainage is thorough. We classify trephining with other forms of scleral puncture. It seems that the trephine opening closes very promptly, so that it is not very different from a scleral puncture. The trouble is, the detachment recurs and the next time it is harder to get back. Until

we know more about the cause of retinal detachment I think we shall be baffled as we have been in the past. The operation recommended by Thompson caused me, when I tried it, the same difficulty that Dr. Green mentions. It is hard to get your needle into that small opening, because it is covered by the conjunctiva and you cannot see it. It seems to me the important thing about the scleral trephine opening is that it does not continue to drain. I should like to know if any of you have positive evidence that it is still draining.

Dr. W. R. Parker, Detroit:—Dr. Lancaster's point, I think, is well taken. Detachment of the retina is merely a symptom and should be treated as such. Any case of detachment coming under one's observation should first be made a good surgical risk, before any operation is performed. In regard to immediate operation in cases of detachment, as suggested by Dr. Green, it has seemed to me it would be necessary to answer two questions: (1) Are we minimizing the possibility of improvement by delaying the operation a reasonable length of time? (2) Do we risk the integrity of the eye by performing the operation early? I believe we are as yet unable to answer either of these questions. The cases carefully studied, after scleral trephining operations, are far too few for us to say whether this operation is to be effective or not. If these cases reported by myself and others are the result of spontaneous cures, I have only to say that in my own experience whatever leads to spontaneous cures has been endemic in the last three years, as before that time I never saw a case of spontaneous reattachment which might be said to be permanent. Now, I don't wish to be misunderstood. I believe they occur occasionally. I am speaking of my own experiences. Out of fifteen cases that I have had I have not increased the number of cures over my last report (which included eleven cases), there being still only three cases with very good results; one case with a brilliant result. The first patient operated on in 1913 had a vision of 4/60, and now after three years his vision is 6/7.5, and you cannot see any signs of detachment.

My desire to determine whether the operative procedure risked the integrity of the eyeball led me to operate with every caution. I have seen no serious reaction following the operation. I have heard of one case which was followed by iridocyclitis. Until the question of danger of delay is answered, it seems to me we ought to be conservative, and not to operate until after rest in bed, pilocarpin, etc., have been thoroughly tried.

Dr. Green (closing discussion):—I certainly appreciate the generous manner in which you have discussed this paper. I presume a good many men have had more or less experience with this operation. Personally, I have not encountered any undue reaction following operation in this very limited group of five cases. I had the privilege of seeing Dr. Wilder operate on one of his patients. He very ingeniously makes his trephine opening underneath the tendon of one of the rectus muscles. I cannot imagine what gave rise to the uveitis in his case. Certainly, it was through no fault of the operative technic.

Dr. Jones apparently regards the subconjunctival injection of cyanid of mercury as useful merely in an antiseptic sense. Is it not possible it may also be useful in stirring up lymphatic activity, thus promoting the restoration of the eye to normal through increased nutritional activity? Dr. Lamb's suggestion is very interesting. Whether or not his view can be established by experimental methods is a question for the future. I agree with Dr. Lancaster that there is no permanency in the opening in the sclera. I believe that it does closes and closes fairly promptly. Whether trephining has any special advantage over scleral puncture, I am not prepared to say. My impression is that the trephine opening does remain

patent longer than a simple scleral puncture. Dr. Parker's observation that patients with detachment should first be made good surgical risks is well taken. My suggestion is that prompt operation, i. e., operation as soon as possible after the detachment, may prove to afford a better chance for eyesight, than late operation, after the failure of nonsurgical methods. In one patient I operated on an old detachment unsuccessfully. In the same patient the operation on the fellow eye within seventy-two hours of the occurrence of the detachment was successful. Had I been privileged to operate the first detachment as promptly, might I not have secured equally as favorable a result?

PRESSURE CHANGES IN THE CURVATURE OF THE CORNEA DUE TO CHALAZION AND OTHER LID TUMORS

T. W. Moore, M.D.
HUNTINGTON, W. VA.

At various times our literature has contained reports of attempts to change the refraction of the eyes by some form of massage or manipulation through the eyelids. These efforts have usually not been sufficiently convincing to justify us in following them; for, while we know that the external coats of the eyeball ordinarily yield somewhat on pressure, and that for every 0.3 mm. of change in the anteroposterior diameter, the refraction is altered by 1 diopter, we also know that when this pressure is removed the eyeball will resume its natural shape, and there will be the same state of refraction that existed before the pressure was applied.

It has been shown that by putting the fingers over the outer canthus and pulling the eyelids outward, the distant vision of low myopia may be improved. This was demonstrated by Mr. E. Donaldson,[1] who also called attention to the fact that this method of stretching the eyelids by the fingers improved the vision in astigmatism against the rule, whether hypermetropic or myopic, and explained that this improvement is due to flattening the cornea in the horizontal meridian.

It has been known for years that persons with eyelids of certain shapes are likely to have definite forms of astigmatism, leading us to conclude that the constant direction of pressure may be a factor in modifying the curvature of the cornea. That chalazion, or other lid tumors may, by their pressure, modify an existing astigmatism, or other error of refraction, has not been noticed in our literature, so far as I am able to learn, except in a note by Henry Dickson Bruns in 1905.[2] However, Oliver[3] does say rather vaguely, I think, "that direct and indirect pressure on the cornea or the sclerotic, as for example, from new growths that are situated either in the orbit or in the eyelids, are said to give rise to corneal astigmatism.

1. The Ophthalmoscope, **2**, 134.
2. Ophth. Rec., **14**, 421.
3. Norris and Oliver: System of Diseases of the Eye, **4**, 429.

Case 1.—In 1904 a patient came to me complaining of poor vision in one eye. Examination revealed on the right upper lid a chalazion, which he declined to have removed. He had been wearing +0.50 cyl. ax. 180 over each eye, but now the right eye accepted —0.50 cyl. ax. 15. This axis was confirmed with the ophthalmometer, but his vision, while 6/6, was not so clearly defined as in the other eye. One month later this patient had the chalazion removed, and shortly after he again complained that his glasses did not give satisfaction. Examination now showed that his refractive error in the right eye was again corrected by +0.50 cyl. ax. 180. The left eye had remained unchanged.

Case 2.—In May of this year, a clergyman, aged 51, who had been my patient for about ten years, came for refraction. He had a large chalazion on the right upper lid. His refraction was R. E. +0.25⊃+0.50 cyl. ax. 130; L. E. +0.75⊃+1.25 cyl. ax. 50. This impressed me at the time; for the manifest hyeropia had been the same in each eye during the years I had had him under observation. I examined his eyes daily for three days, always obtaining the same result. At this time I obtained his consent to remove the chalazion. One week later his refraction was R. E. +0.75 Sp.⊃+0.50 cyl. ax. 130. L. E. +0.75 Sp. ⊃+1.25 cyl. ax. 50. This showed the manifest hyperopia to be the same in each eye that it had been during previous years.

Dr. Bruns' observations were on himself and are so complete that I will quote them in full:

Case 3.—In 1887 the refraction of the writer, then 22 years of age, was most carefully measured under repeated instillations of homatropin, by Dr. Howard F. Hansell of Philadelphia; it was found to be R. +0.50⊃+0.50 cyl. ax. 90. L. +0.25 Sp. ⊃+0.50 cyl. ax. 90. V. was 20/xv with each eye, and has always so remained. April 8, 1899, the astigmatism measured with the J. S. ophthalmometer was found to be R. E. 0.50 ax. 105. Feb. 3, 1901, the same instrument gave an identical reading. In May, 1904, a small chalazion, about 3 or 4 mm. in diameter, developed with moderate inflammation, in the upper lid of the right eye. At the same time the vision of this eye became uncomfortably dim at all distances. R. E. V. 20/xxx, the outlines not clear cut, and while Sn. No. 4 could be read with the presbiopic correction, +1.75 Sp. ⊃+0.50 cyl. ax. 90, the print looked smeared and not as it appeared through the same glass with the left eye. Now, on May 27, 1904, very careful measurement with the ophthalmometer showed R. E. O.; L. E. 0.50 ax. 105, and R. E. +0.50⊃+0.50 cyl. ax. 165=20/xx. This condition lasted a month or two, when the chalazion began to disappear and gradually, very gradually, the vision for near

became clear and comfortable with old correction, and V. R. E. 20/xv. Today (April, 1905) the ophthalmometer shows in R. E. 0.50 ax. 75 once more."

A very interesting feature is that in the three cases reported the amount of hyperopic, or hyperopic astigmatism, was diminished, showing that these changes are due to a modification of the corneal curvature and not to a shortening of the eyeball in its anteroposterior diameter which would increase the hyperopic condition.

Conclusion.—I believe that the three cases cited prove conclusively that these neoplasms may cause changes in refraction that will subside with the removal of the tumor. In prescribing lenses for patients suffering with these conditions, it is well to bear this in mind and to tell the patient of it. Especially is this true when it is under these circumstances that we first prescribe lenses.

DISCUSSION

Dr. J. M. Ray, Louisville, Ky.: The observations of Dr. Moore corroborate the observations I made some years ago. A case very similar to those reported by Dr. Moore presented itself. There was a large chalazion. When I looked at the record made on this patient at the former visit it did not correspond with the present record. On measurement the astigmatism was quite changed. After removal of the chalazion, some months later, there was again a change corresponding more nearly with the first record.

I have frequently seen cases where pressure on the lid would change the astigmatism. I have a friend who, by squeezing the lid, makes a difference of 1 diopter while he is being observed with the Javal ophthalmometer. There is no question that growths on the lid will temporarily change the curve of the cornea and must be taken into consideration in measuring the refraction.

DEVIATION UPWARD OF THE ONE EYE, DUE TO OVERACTION OF THE INFERIOR OBLIQUE, CONSECUTIVE TO CONGENITAL PARESIS (OR INSUFFICIENCY) OF THE SUPERIOR RECTUS OF THE OTHER EYE

ILLUSTRATED BY MOVING PICTURES

FRANK C. TODD, M.D.
MINNEAPOLIS, MINN.

CASE 1.—On July 13, 1916, I was consulted by Bertha K., aged 13, who gave the following history: Deviation of the eyes was first noticed when patient was 6 weeks old. It was then not constant, but appeared when she looked in certain directions. It became increasingly noticeable. She was fitted with glasses when 5 years of age, and wore them three years without correction of the deviation.

In answer to inquiry, patient stated that she was not bothered with double vision, except when her vision was directed toward a near object (when convergence was required). But the mother stated that she was continually running into things, particularly when they were on the right side, and that the family had observed that the head always assumed a certain position, viz: a tilting to the left and a turning toward the left. The parents had, however, not associated the head position with the eye defect.

Examination:

Versions	Up	Down	Out	In	Vision
Right	60	30	50	65	R.. —6/9 —6/6 + 2.00
Left	50	45	40	65	L.. —6/5 —6/5 + 0.50 ⊃ + 0.50 ax. 90

It will be observed on looking at the moving picture that when the patient was asked to fix the eyes on the camera and turn the head toward the left, the eyes were parallel; then, as she slowly moved the head toward the right, while the left eye continued to fix, the right eye turned upward; and, as related to the left eye, it deviated somewhat outward.

The pictures shown in the illustrations herewith, numbered 1 to 10 inclusive, demonstrate the movements of the eyes from extreme right to extreme left.

We found that the deviation upward amounted to about 25 degrees, and outward to a maximum of about 15 degrees. Then, if the patient was asked, while the head was in this position, to fix with the right eye, the left eye would drop downward and outward to a corresponding degree. The patient has partial suppression of the image, so that tests could not be made with the Maddox rod, and had to be made with the cover test.

These illustrations show the continually increasing deformity of the right eye, caused by overaction of the inferior oblique as the patient looks from the extreme right to the extreme left.

Note the gradual upturning of the right eye, and that while there is an apparent convergent strabismus, in reality as related to the other eye it is divergent.

The pictures shown in the illustrations in this article show the patient looking in various directions, all the way from the extreme right to the extreme left, while the moving pictures show the patient turning her head in various directions, but the effect is the same.

Diagnosis.—This is a case of congenital insufficiency (paresis or muscle anomaly) of the left superior rectus, and secondary deviation upward of the right eye, caused by compensatory overaction of the inferior oblique. Tilting of the head produced parallelism and consequently the head almost continually assumed this position.

Operation.—On July 21, a complete myotomy of the right inferior oblique, according to the method described by Duane, was performed under general anesthesia. On August 2, measurements showed some improvement, but still considerable deviation. On August 3, under local anesthesia, the conjunctiva was

After operations; extreme rotation to the right.

After operations; extreme rotation to the left.

re-opened under the right eye, and an exploration made to determine to a certainty whether or not there were any remaining attachments. It was found that the inferior oblique had been completely severed. The superior rectus of the left eye (the paretic or insufficient muscle) was advanced by a tucking operation. The results from this operation proved satisfactory, and parallelism was secured, while the tilting of the head was completely relieved. The patient was allowed to go home on August 15, promising to return again in a month. I have been unable to get the patient to return, but have received a letter from her dated November 27, in which she says, "I do not see double, and my eyes are straight." (See the two larger photos taken several months after the last operation.)

This condition was called attention to by Dr. Duane in a paper published by him in the *Annals of Ophthalmology*, October, 1904, and again in an article reporting a case in the *Archives of Ophthalmology* in 1916, in which Dr. Duane reports having performed tenotomy of the inferior oblique for this condition with relief in several instances, and states that he had seen about forty cases, and therein he describes his method of doing the operation.*

I did not get the relief in my case until I had advanced the paretic muscle, and believe that in the next case I shall attempt the advancement of the paretic muscle before cutting the inferior oblique.

In answer to my letter to Dr. Duane giving a description of this case, he writes that his associate, Dr. White, has operated on seven of such cases, and Dr. Knapp on one; that he himself has operated on eight or nine, doing a tenotomy of the inferior oblique with success in all but one case, and suggests the possibility that his one case and mine were alike, and explainable on the basis of some anomaly of insertion. In my operation for advancement, I noted no anomalous attachment of the tendon to the globe.

I present this case because it is evident that such cases are not infrequent. They are, however, evidently often unrecognized. The moving picture will make this so evident that one cannot fail to diagnose the condition.

The characteristic symptom which may be first evident to the friends is the tilting of the head, although in some cases a secondary lateral (either divergent or convergent) strabismus may result. To quote from Dr. Duane: "When head-tilting fails to give relief, and partial or complete suppression has not occurred, the patient often involuntarily adopts the expedient of diverging or converging the eyes, until the double images are so far apart as to be no longer confusing. In this way, at the age of 2 or 3, or perhaps later, a squint develops. Of the cases of acquired convergent and divergent strabismus that I have observed, quite a fair proportion have developed in this way, the acquired lateral squint being secondary to the congenital vertical deviation."

In making the examination, it may be found that the patient may fix with either eye, but in the case of an insufficiency of the superior rectus of the left eye, and a consecutive overaction of the right inferior oblique, when the object looked at is carried

* Dr. Posey, at a later date, presented a paper before the American Ophthalmological Society, on "Tenotomy of the Inferior Oblique."

so far to the left as to be hidden from the right eye by the nose, and at the same time is carried somewhat up—the left eye, which now has to fix, would tend to lag down, and the right eye would tend to shoot obliquely up.

"A final proof that the upshoot of the right rotation was due to spasm of the inferior oblique and not to any peculiarity in the insertion of the internus, would be afforded by the fact that when the eyes converged strongly, the right eye, although turned strongly in, does not shoot up, but does so at once when *both* eyes are turned to the left."—(Duane).

If the case is one of paresis of the right inferior rectus with consecutive overaction of the left inferior oblique, the effect would of course be the same, but the various faulty movements described would be transferred to the other eye.

In case binocular vision is present, tests may be made with the red glass and the tangent screen of Duane.

These cases should be operated on early, thus obviating the disfiguring wry-neck or strabismus, which may otherwise result.

DISCUSSION

Dr. Wendell Reber, Philadelphia: I think our thanks are due Dr. Todd for taking this unusual method of showing this, which is the only way it could have been shown. The frequency of these cases is greater than generally supposed. Of this I feel sure from the number of cases reported by Duane and Posey, and I have seen eight cases—seven I am perfectly sure about. On one I did a tenotomy of the inferior oblique and advanced the superior rectus on the good eye with perfect results. As a rule, the head is tilted, and I think such patients should be operated on comparatively early. The interesting thing is that they fix with the perfect eye, and that is what produces the tremendous upward rotation when the eye is turned. We seem to lose sight of the fact that when the eyes are rolled up both eyes are stimulated. To be sure, the inferior obliques also enter into the matter, and sometimes you get a pure upward rotation, as in the picture I show here. These cases are divergent strabismus and are due to a congenital defect. Where you have to deal with these cases I think the ophthalmologist will be safe in doing the advancement on the superior rectus. (Illustrates operation on board.)

Dr. John Green, Jr., St. Louis: I recently operated on a boy of 6 years, who had a slight internal squint associated with upward deviation of the left eye, with compensatory head tilting. The deformity was by no means as conspicuous as in Dr. Todd's case. Tenotomy of the inferior oblique brought the visual axes nearly to parallelism and entirely abolished the head tilting. My impression is that in low degrees of this anomaly simple tenotomy of the inferior oblique will be all sufficient.

Dr. Frank Todd, Minneapolis (closing discussion): I am abliged to Dr. Reber for calling attention to these points. The reason I brought this before the society is because I felt the condition, while relatively infrequent, is seen rather frequently and remains unrecognized. We are likely to consider these as cases of lateral strabismus and do the wrong operation. The diagnosis is very simple if we keep in mind the movements shown in this moving picture and described in this paper.

THE USE OF EYES OF KITTENS IN OPHTHALMIC OPERATIVE TEACHING

W. A. Fisher, M.D.
CHICAGO

Teaching institutions do not encounter any great difficulty in the instruction of nose and throat operative technic, sufficient clinical material being always available and the procedures being usually of such nature as to make it possible for a novice to undertake them, under the direction of a capable assistant, after sufficient preliminary reading and instruction. Such, however, has not been the case in the matter of ophthalmic operative teaching. To begin with, there is not a great deal of ophthalmic surgery to be done, and the majority of the procedures are so pregnant with such grave possibilities as to preclude their being intrusted to one who has not had experience.

Clearly this experience must be gained on the eyes of the lower animals, but up to the present time no great amount of useful work has been reported in this field. Sheep's eyes have been used for practice in muscle operations, and those of the pig for operations upon the eyeball; but neither are satisfactory. In the sheep the capsule of Tenon is so poorly differentiated and so many accessory tendonous attachments are encountered as to make the work atypical. As obtained commercially, pig's eyes are delivered enucleated, thus precluding practice on muscles; and since they have such a large and thick cornea, are quite impractical for corneal work. The iris, too, is so thick that operations on it are likewise very unsatisfactory.

Dissatisfaction with the use of the above and realization of the need of something better—the use of an eye more nearly approaching the condition obtaining in the human—has induced me to conduct a series of experiments in an effort to find a more satisfactory substitute. Since the greater portion of ophthalmic surgery is concerned with the cornea or iris, alone or combined, my efforts have been directed to finding material in which these structures are analagous to those of the human and have included practically every available small or domestic animal.

The eyes of guinea pigs were rejected because of the small size of the cornea, even in well-grown specimens. Those of

the rabbit are nearly like that of the human in point of texture, but the anterior chamber is so shallow that it is quite impossible to make the incision for iridectomies or cataract operations. The eyes of domestic fowls were rejected because of the cartilaginous character of the limbus which effectually prevents proper puncture and counterpuncture, and makes it impossible to express the lens by means of pressure.

For muscle work the eyes of the dog were found far superior to those of the sheep, because of the well marked Tenon's capsule and the character of the tendons, which are the same as in the human eye. It is necessary, however, to select the eyes of well-grown animals to find muscles of a size comparable to those in the human subject. This, as in the sheep, results in the selection of materal in which the cornea is too large and dense for satisfactory work on the globe.

For work on the eyeball proper, the eyes of kittens were found so far superior for all purposes to all the others mentioned that, once used, they will never be discarded. The reason for this lies in the fact that, while, as in other lower animals, the ratio of diameter of cornea to size of the eye is greater than in the human, specimens may be readily obtained by proper selection, in which the size and thickness of cornea, depth of anterior chamber and texture of iris — the important points of this work—are analagous in every respect to those of the human eye. To meet these conditions, one should use the eyes of kittens from 4 to 5 weeks old and weighing about twenty-four ounces.

The question of the availability of material is not at all pertinent, because no difficulty need be experienced, even in our larger cities, in procuring it regularly in quantity and at really very small expense.

Technic.—The following has been found to be a good working plan: The kittens, after having been dusted lightly with powdered naphthalin to dispose of any vermin present, should be killed before beginning work. The killing may be most conveniently and cheaply done by the injection of one-half grain of strychnin sulphate intrapleurally. Cut away the lids and nictitating membrane, which interfere and the absence of which does not impair the work in the least. Then do tenotomies on the recti muscles, and finally, since the eye, because of its deep-set position, must be removed and placed in a mask for cataract work, enucleate by any of the methods advised.

The eye should now be fixed in a suitable mask, which can be easily improvised from a deep cigar box. (Fig. 1.) The bottom of the cigar box being too thin, the lid is removed and fastened securely to the bottom by means of glue or nails. Locate two points on the re-inforced bottom three-fourths of an inch from either side and one and one-half inches from one end. At these points burn holes with an ordinary hot poker not to exceed one-quarter of an inch in diameter and cautiously enlarge them to one-half of an inch, taking care not to burn too deeply into the wood.

Strive to make a concavity in which to have the eye rest snugly and at the same time have enough of the bottom of the box behind it to prevent its drawing through when in position.

Fig. 1.—Cigar Box Mask.

Drill two more holes large enough to accommodate any small wooden cylindrical rod—a lead pencil does nicely—near the free edges and opposite those in the bottom of the box. Slot one of the holes and make a key to fit the slot snugly that when pressed home it will prevent the pencil's turning. Flatten one or two sides of the pencil and the mask is ready for use as follows:

Pass a strong, sufficiently long thread into the scleral tissue near the optic nerve and drop the ends through one of the bottom holes, either right or left, as may be desired. Make one or two overlapping retaining turns over the flattened pencil and then continue turning until the eye is drawn snugly into the cup made for it at just the proper tension as determined by the tonometer.

For this special work I have devised a new mask (Fig. 2), with which the eye is similarly secured and maintained at the proper tension and which have some advantages over the mask

made from the cigar box, as there is less trouble in passing the thread.

With the eye thus fixed, the regular cataract procedure may be taken up in order, viz: Incision, iridectomy, by any of the described methods; capsulotomy with cystitome or capsule forceps, if practice in the classical method of extraction is desired; lens extraction in capsule and, finally, replacement of the iris and toilet of the corneal wound.

We must admit that experience is our greatest teacher in this operation, but that material is practically unavailable. Eyes are too precious for teachers to allow students to operate;

Fig. 2.—Improved Mask in Use.

hence, the only chance one has to acquire experience in this delicate surgery is to witness operations.

If a student could have an opportunity to operate for cataract on 100 eyes and this experience could be obtained in a short time, he could acquire quite a good technic; but, this experience extending over many years, as is usually the case, by the time one has acquired a reasonably safe technic he is loath to change or take up another method. If, then, one will operate on a sufficient number of kittens' eyes to get a technic, he can remove lenses in capsule quite as well as by the old method. A trial of the method will convince any one of its efficacy.

There are probably few operators who would not accept the intracapsular operation, provided they could have enough

experience to master that technic. A good incision is imperative whatever method is selected for a cataract operation, and this can be mastered provided enough material is used. In addition to the above, all of the operations on the eyeball that are described in the textbooks may be performed on these eyes precisely as they would be in the human subject, with or without preliminary enucleation, as the student may elect.

The reason that ophthalmic surgery is generally considered difficult lies in the fact that all, save our most experienced operators, approach a given case with a feeling of unpreparedness and uncertainty, resulting from a lack of the co-ordination of brain and hand that can come only from operating many times.

With the material described, however, this need not be the case, for in a comparatively short time, which is a distinct advantage in acquiring technic, any student can with, say 100 kittens, do as much operative work as the busiest ophthalmologist in many years. One hundred kittens (200 eyes) will furnish material for 400 muscle operations, 200 enucleations, 200 iridectomies, and 200 cataract extractions, making in all, 1,000 operations. If the student uses this or twice this amount of material, he cannot fail to bring to his operating room an experience and confidence that can be secured in no other way.

WHEN AND HOW SHOULD A CATARACT OPERATION BE PERFORMED?

The majority of the world's ophthalmic surgeons would say to the patient, "Wait till you are blind or the cataract is mature and have the operation"—that is practiced by most ophthalmic surgeons.

It is a serious matter to advise a patient with immature cataract to delay the operation when his adviser knows this advice would not be given if he had material enough to master the intracapsular technic. Smith of India states that when the lens becomes so opaque that vision is no longer useful for ordinary practical purposes, it should be extracted in capsule.

Ophthalmic surgeons the world over would give the same opinion if they could remove lenses as Smith does, and the only excuse is the want of material to operate on.

Removing a lens by any method is a delicate procedure and requires much experience to become proficient. Whichever method the operator received his experience in is the one he is familiar with, and when he has operated about fifty or more times, he is loath to change from the method he feels that he has mastered.

Cataract operations by any method performed by fair operators on patients commonly called good are usually successful, and those that are failures would usually have been successful, provided no accident or complication had occurred. Accidents and complications must then be emphasized more than the method of operating. The method of operating should be selected that will give the best general visual results, and the complications common to this method should be emphasized.

If the lens is successfully removed in capsule, inflammation is rare, and it is not necessary to make a secondary operation on the capsule. If this method can be successfully mastered, these advantages, together with the fact that the operation can be performed at any stage of opacity, are surely worth consideration.

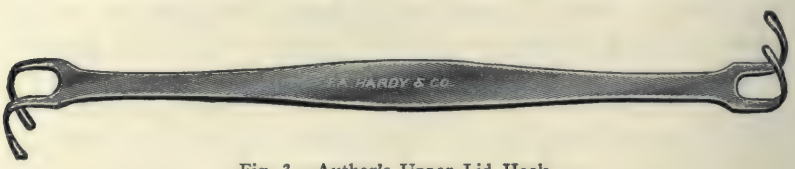


Fig. 3.—Author's Upper Lid Hook.

The greatest objection to the intracapsular cataract operation seems to be the fear of loss of vitreous. This complication is recognized as serious when operating by any method, but when it occurs before the lens is delivered, it is more serious; because the lens must be delivered, and this cannot be accomplished by pressure on the cornea, and if the lens is delivered with a wire loop passed into the vitreous, the complication is very grave. This accident must be emphasized and, if possible, a method of taking care of it devised.

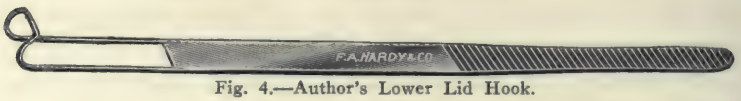


Fig. 4.—Author's Lower Lid Hook.

How to Avoid Complications.—The operator should familiarize himself with the hooks, not on cataract patients, but on anyone with the eye anesthetized by holocain. If the lids are mastered in this manner by the operator, he will have little trouble in instructing an assistant or nurse. The one thing necessary for proficiency is enough practice on human eyes.

When operating by any method the lids must be held away from the eyeball, and this is best done by the lid hooks I have devised (Figs. 3 and 4), in the manner described in Figures 5 and 6, or by those devised by Dr. Derrick T. Vail.

Loss of Vitreous Preceding Lens Delivery.—This complication is a serious one unless handled in a proper manner. Figure 7 represents Smith's method of removing the lens when the intracapsular operation is being performed and a loss of vitreous is imminent or has actually occurred.

Fig. 5.—Author's double lid hook, holding the upper lid away from the eye, keeping all pressure from the globe, the assistant's fingers resting upon the patient's nose. The thumb of left hand holding the lower lid down, a second assistant or nurse holding the brow up. The assistant holding the lid hooks stands at the left side of the patient when operating upon either eye.

Fig. 6.—The same as No. 5, except the double hook is pulling the lid forward or down to take off all pressure in impending or actual loss of vitreous, the double hook being depressed to keep from slipping out from under the lid.

Delivery of the lens by the intracapsular method cannot be successfully demonstrated on kittens' eyes, because the animal is too young, but experience can be obtained in the use of the Smith spoon and the author's needle. If this method is emphasized and plenty of kittens sacrificed, the complication that seems to keep the intracapsular operation from being accepted

will not be as serious as it now appears. Many operators have attempted the intracapsular operation without this experience and have failed.

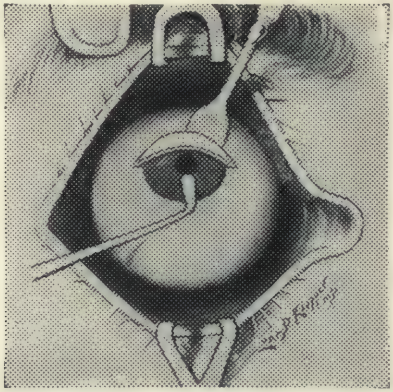


Fig. 7.—Smith's Method with Author's Modification of Operating When Vitreous Precedes Lens Delivery: If loss of vitreous preceding lens delivery has occurred in the intracapsular operation, the lens is delivered by the Smith method with the lids held away from the globe by author's retractors, the same as in the beginning of the operation. Pressure of the hook is removed, the Smith spoon is passed down behind the lens and pressure is then made to cause it to slide up the spoon and out.

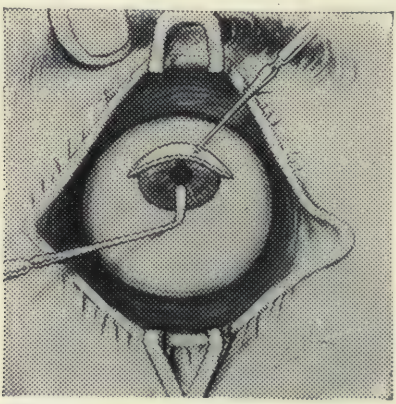


Fig. 8.—Author's Method of Operating When Vitreous Precedes Lens Delivery: Loss of vitreous preceding lens delivery has occurred in the intracapsular operation, the lids are being held away from the globe by author's lid hooks, the same as when the operation began. The author's needle which was held in the left hand waiting for such an accident, is stuck into the edge of the lens, and, with just a little pull upward, together with slight pressure upon the cornea with the hook, the lens either comes out in capsule, or the capsule ruptures.

The technic of the cataract operation as described will spell success no matter what method is selected. It seems strange that many operators of large experience advise mastering the capsulotomy method and operating not less than 100 times before taking up the intracapsular operation, whereas emphasis

should be given the complications of cataract operations by any method.

How to Proceed to Deliver a Lens.—When an operator can make a good incision on kittens' eyes, has mastered the lid hooks on human eyes, Smith spoon and author's needle on kittens' eyes, he begins the operation by making as good an incision as he can, makes the best iridectomy he can, and instead of cutting the capsule he proceeds to remove the lens by the intracapsular method, holding the needle in position as described in Figure 8, ready to stick into the lens if it refuses to be born after he has made pressure he considers safe, or if vitreous loss seems imminent, or, if it has actually escaped. The spoon is reserved for any case when the lens slips back into the eye, preventing the use of the needle. The more expert the operator, the less use he will have for the spoon or needle.

Some capsules will rupture when the needle is used, but most operators would prefer a secondary needling to a loss of vitreous.

Fig. 9.—Smith-Fisher Instrument.

After-Treatment of Cataract.—If the lens is removed in its capsule, inflammation is rare and the daily treatment recommended after the capsulotomy method is omitted. The bandage can be changed the fourth day and a new one applied, but the lids should not be opened, and this bandage should not be disturbed for five days more, unless the patient complains. Nine days after the operation the corneal wound is usually closed, the anterior chamber reformed, the bandage omitted, and the patient is given smoked glasses. If a prolapse of the iris is found at the first inspection nine days after the operation, the eye is rebandaged for four more days, at which time the prolapsed iris is cut off. After a cataract operation the patient should be put to bed and kept there twenty-four hours, after which he can be up or down as is most comfortable, advising him that the bed is the best place for the first week. Daily inspection of the eyes is unnecessary and often harmful.

Conclusions.—(1) Master the lid hooks and teach an assistant by practice on human eyes, and the incision, Smith spoon and author's needle on kittens' eyes before beginning a cataract operation by any method.

(2) Students cannot become competent to do surgical operations on the eye by watching others.

(3) The eyes of 4 weeks old kittens are better for teaching ophthalmic surgery than are those of other animals.

(4) Any student who has prepared himself for ophthalmology can get an operative technic in this manner not equaled by any other.

DISCUSSION

Dr. Patton, Omaha: I believe we all sometimes feel we could best serve our patients with a Smith operation, but realize it demands technic which we have not mastered. I was so fortunate a few weeks ago as to see Dr. Fisher demonstrate this new eye holder and his experimental work on kittens. I have sacrificed several kittens' eyes in the interest of science, and have been pleased with the assurance it gave me in the expression of the lenses by this method, without the loss of vitreous, which we all dread. Not only does it give us experience with the technic, but also familiarizes us with the special devices of Smith and Fisher. I enthusiastically endorse what Dr. Fisher has said. After we have acquired this technic on these kittens, I am sure we shall use this method in the cases where it is indicated.

Dr. Banister, Omaha: Why is it necessary to kill these kittens with strychnin? There are terrible convulsions and great suffering so caused. Why not use a few drops of chloroform, which will produce a rapid and painless death?

Dr. J. W. Millette, Dayton, Ohio: Dr. Fisher's method gives an opportunity for practice that one seldom is able to get otherwise. My first experience was gained on living dogs. To me it seems better to work on the eye of a living animal with the globe in its natural bed and with its natural attachments. We wrapped the dog in a gunny sack, fastened his legs down to his body, put a strap around his nose and mouth, and then cocainized the eye. An assistant held the dog and the operation proceeded as on the human subject. The eyes were allowed to go without bandaging, and those I kept under observation healed rapidly without accident or infection. I see no reason why kittens might not be used just as effectively as dogs. The surgeon who does a cataract operation only occasionally will find this practice almost necessary, and if he does the intracapsular operation he must have practice, practice, plenty of practice.

Dr. F. G. Steuber, Lima, Ohio: Many of us with limited experience have had to do more or less work on the cadaver and pigs' eyes, which are fairly satisfactory for the work.

The muscle work, also the Smith cataract operation, no doubt may be quite satisfactory on cats' eyes, though I do not quite understand how it works out when treating of pupil and iris. Unlike the human eye, the pupil is elliptical in the vertical, or nearly so, which applies to the felidae or cat tribe in general. However, this may be no objection. If so, perhaps the essayist can explain.

Dr. Fisher (closing discussion): At least twenty members of the Society have asked me within the last two days how to get to India and the best time of the year. Smith's time for retirement from the Indian Medical Service is past, and he will probably not be in position to give others an opportunity to become proficient under his care. While many

in India are operating by his method, they will not be likely to be as generous as he has been, because they, like us, want the experience. I am glad Dr. Patton has had some experience with kittens' eyes and feels so kindly toward the method.

Regarding Chloroform.—It is expensive and not so sure as the injection of strychnin. One-half grain injected into the pleura insures results in less than one minute, and the operation seems painless.

Dr. Steuber asked about the iris. It is about the same structure as the human eye, and while the pupil seems oblong in the live kitten, it is round and dilated when the kitten is dead. When the corneal incision is made, which seems about the same as in the human, the iris is found to be a delicate structure, and the practice of making an iridectomy for cataract or glaucoma is very much like it is in the human.

The technic of the incision, iridectomy, and lens delivery can be obtained upon the kitten in some respects better than on the human, especially because there is no responsibility and one can do the work deliberately.

TREATMENT OF CATARACT AT THE CENTRAL BRANCH OF THE NATIONAL MILITARY HOME

J. W. Millette, M.D., F.A.C.S.
DAYTON, OHIO

The patients we have to deal with at the National Military Home are not like those met with in private practice. They are men whose ages are between 70 and 85 years; men who most frequently have no home ties or close relationships; men who, as a rule, have lived lives of hardship and exposure. Many have been none too careful in the kind of life they have lived; some are actual derelicts; many are cripples; most are in disease; few indeed could be considered healthy.

Perhaps the most frequent and troublesome condition to be met with in the after care of these patients is some genito-urinary trouble, nephritis, cystitis, or acute retention of urine. Together with their advanced age and low general physical condition, their mental condition is not of the best, and post-operative insanity must quite frequently be taken into account. Chronic nasal catarrh and so-called senile conjunctivitis are common and need to be taken care of.

It must be remembered that these men are the remnant of that great body of men who fifty-two to fifty-five years ago took part in our great internecine struggle, and were exposed to all the ills, mental, physical, and moral, which such a life must of necessity entail.

Our first care always is to get the patient into as good condition as we can. As might be expected, some will brace up after a few days of rest and treatment, but a goodly number does not. The patient is taken into the hospital several days before the operation is scheduled. The intestinal tract is carefully cleansed, the urine is examined one, two, or more times as conditions indicate. Usually a complete history of the patient as to genito-urinary trouble, or heart trouble, or, in fact, any chronic functional difficulty, comes with him to the eye wards. The blood pressure is always taken, and, if abnormally high, an attempt is made to lower it. However, high blood pressure is not a grave contra-indication to an operation. I have operated several times with no evil results when the sphygmomanometer

registered between 200 and 235 mm. of mercury. The only difference noticed was a slightly longer time required for healing.

A very annoying condition and quite a common one among these old men is a subacute catarrhal conjunctivitis. Examination of the secretions from these eyes very seldom shows any virulent organism present, but only just such as are found in the secretions of most old peoples' eyes, and yet there is a discharge which gathers at the roots of the cilia and at the inner canthi and which will soil the test bandage. A zinc sulphate astringent or a 1 per cent. solution of silver nitrate has been most efficacious in treating these cases.

Commonly associated with this is a chronic rhinitis, which is treated by an alkaline spray and menthol vapor.

Every organ of the man is looked after carefully and the general condition raised to as high a degree of efficiency as possible before operating.

An intracapsular operation has been performed with very few exceptions. A brief description of this procedure will probably be in place at this time.

The instruments used are those used and advocated by Colonel Smith of Amritsar, India. The site of the operation is prepared after the usual manner, and the patient is then brought to the operating room in his bed. At the Home all operations on the eye are performed in the bed, which is equipped with large castors. This equipment was furnished at the request and under the direction of the late Dr. D. W. Greene. The bandage being removed, the eye is anesthetized by instilling a drop or two of a 4 per cent. solution of cocain four times within about ten minutes. The conjunctival sac is then thoroughly flushed after the manner advocated by Colonel Smith. The special speculum being introduced, it is possible to lift the lids away from the globe, so as to freely expose the entire conjunctival sac, which is flushed with a warm, sterile, normal salt solution. Dr. Greene used a 1:3000 bichlorid solution for a time, but abandoned it because it appeared to excite an undue conjunctival inflammation. I have continued the use of the normal salt solution.

It is our custom to leave the speculum in during the incision and the iridectomy. The incision is made wholly corneal, including fully half the circumference; the puncture and counterpuncture entering and emerging respectively a milli-

meter or more external to the corneoscleral ring. If the tension is low or there is indication of a fluid vitreous, the incision is brought out high on the cornea; otherwise near the corneoscleral margin. When this high incision is made, it seems a trifle more difficult to deliver the lens, but it very much lessens the danger of loss of vitreous.

An iridectomy is always made (I have not tried to do an intracapsular operation without making an iridectomy), sometimes expressing the iris with the forceps without entering the wound, and again after the usual method. I have failed to see any difference in the results. The speculum is then removed and the assistant holds the upper lid with the special hook in his right hand, and controls the lower lid with his left thumb or first finger. The operator stands at the top of the patient's head, his left arm under the right arm of the assistant. In his left hand he holds the double-ended spatula, the small end of which he has always ready for use in case the vitreous presents or the lens refuses to start. In his right hand he has the delivery hook, the ball of which is placed on the cornea, a millimeter or so below a point immediately over the center of the lens. The arm of the hook is laid flat on the cornea.

The delivery of the lens is quite impossible to describe accurately. One must not only see it done, but *do it* in order to appreciate it, and yet in a general way it is as follows: Having placed the hook as above described, a *quick* gentle pressure is made directly backward toward the posterior pole of the globe. This should loosen the zonula sufficiently, so that by continuous pressure the lens will present in the corneal wound. The direction of the pressure from this point on should follow the lens in such a manner as to facilitate its emergence. If the lens has been more freely loosened at the lower edge by the first sharp pressure, it may not immediately present in the corneal wound, in which case the ball of the hook should be moved lower on the cornea, and the lens will then turn over and the lower edge emerge first through the wound—the so-called tumbler. If the lens does present, but seems to stick in the wound, I do not hesitate to tease it by changing the position of the hook on the cornea and by changing the direction of the pressure.

The lens delivered, the pillars of the iris are replaced and the edges of the wound coapted as perfectly as possible. The lids are closed and covered with sterile vaselin. A light cotton

pad is placed over both eyes and fixed with adhesive strips, over this on the operated eye is placed an aluminum shield. A cotton pad is placed across both eyes and held on with a bandage. The patient is then kept quiet on his back until the next morning.

I have not thought it wise to discuss any of the complications which may occur during the operation, as that would make the paper too long. Besides, the one reason for presenting this paper is to get your experience with and opinions of the open treatment after cataract operations.

Having observed for a number of years the frequency of postoperative mental disturbances and the many even serious kidney and bladder troubles, we determined to try out a less severe regimen of bandaging and much less restraint of activity of the body, and have since continued this plan, lessening the tension whenever we felt it would be an advantage. It is our custom to operate about 1 o'clock in the afternoon. As stated above, the patient is kept quiet as possible until the next morning, when the bandage is removed and the eye examined. There being no unusual appearance, a very light pad is put over the operated eye. The patient is allowed to turn to the side opposite the operated eye, or if he complains of his back hurting, we place a back rest in the bed for him and he sits up. The use of atropin is not routine. Drugs are used only as indicated. For protection, particularly from the patient's fingers while he sleeps, the nurse places at night an aluminum shield over the pad on the operated eye. On the second morning the dressing is removed and, there being no contra-indication, is not replaced. From this time on he has the freedom of both eyes, except at night, when the light pad and aluminum shield are used for protection as long as may be thought best. The patient is allowed to sit in a chair on the second day. About the only restraint from this on is, that we keep the patient in a darkened room for five days or more.

Under the conscientious and efficient care of Dr. John E. Kelly, the resident assistant, who has worked persistently and enthusiastically with me during the past three years, and more than a year before than with Dr. Greene, and the exquisite care of the trained nurse, Miss Anna D. Westcott, who has served in these eye wards more than thirteen years, this method has proven so much better for all concerned, that we are convinced that with the conditions under which we must work, it by far excels the closed treatment. And why not? Bandage both

your eyes, lie on your back for twenty-four to forty-eight hours without moving and, though you do not have the worry incident to an operation, I am certain you will have to admit the very great nervous strain it entails.

The lids form the most perfect bandage and protection to the eye, and it is only necessary to keep the operated eye closed until the first intention of healing has progressed far enough to allow the movement of the edge of the lid over the wound without injury to it. This occurs most frequently after eighteen to twenty hours.

That the danger of infection is lessened is equally true, we believe. Given germs and a proper soil—then darkness and absence of air, together with heat and moisture, are unquestionably the most favorable conditions for reproduction. The converse is obviously true. With the bandage we add darkness, exclude air and retain heat and moisture and thereby make a most efficient incubator. The absence of a bandage facilitates the examination and treatment of the eye, should that become necessary.

The natural conformity of the lids to the shape of the eyeball molds the coapted edges of the wound into the normal curvature of the cornea, and this, I believe, tends to obviate cicatricial astigmatism, while a bandage would probably increase it.

In closing let me say: I have indicated the class of patients, the pre-operative care, and the manner of operating, for the sole purpose of bringing to your notice an open method of post-operative treatment. This method is based on my experience in a few more than 200 cataract operations, performed at the National Military Home, in St. Elizabeth Hospital, and my private practice and which covers a period of a little more than three years.

DISCUSSION

Dr. W. A. Fisher, Chicago: I want to congratulate Dr. Millette not only on his work, but for the pluck of telling us about it, because many would not be bold enough to come before this Society with radical changes. The Society will be benefited by such papers. It has been my rule for the past three years to leave the bandage on for nine days, but I believe the bandage is not of so much importance as a good operation.

If the lens is removed in its capsule, treatment is usually unnecessary, and if the patient can have as good results by Dr. Millette's method as could be obtained by longer periods of bandage, his method should have the preference. I believe the bandage can be omitted from the fellow eye after the second day and the patient given his freedom, because the corneal wound is usually closed before that time, but I am inclined to favor noninterference for nine days with the eye operated on, provided the patient does not complain.

About ten years ago Dr. Scales read a paper before the American Medical Association on the open treatment as practiced by Hess. I tried the method on one case and the result was perfect. Like many at that time, I did not feel justified in trying a new treatment, because I felt that the textbooks must be right.

The open treatment is not an experiment with Dr. Millette. It is a fact based on a very large number of cases and deserves very careful and favorable consideration.

Dr. W. B. Lancaster, Boston: It has been my practice for some time to remove the bandage the second or third day in the great majority of cataract operations. A Ring's mask is at once applied, because it is dangerous to leave the eye unprotected. When you are willing to allow the patient to see a little, make a small hole in the mask over the fellow eye, usually about the fourth or fifth day.

The discomfort of lying quietly in bed for the first twenty-four hours can be greatly reduced by the proper use of drugs. The patient should be given a sedative before and after the operation, so as to "rob surgery of its terrors.'

One advantage of the Ring's mask is that you do not need to have the room dark. The patient is cautioned not to open the eyes widely, not to look down, and not to squeeze the lids forcibly when closing the eyes.

Dr. Wendell Reber, Philadelphia: Dr. Parker is always modest and I think I am more so, for I think I have done about 500. I believe in chemical restraint for these people, and I have used bromid and chloral one or two hours before the operation and long enough after to keep them nicely quiet. I use a light dressing. I find patients will tamper with their eyes. I believe in the metal mask, but not in the bandage. I believe in mercury and salicylates before the operation and after it. I do not believe in taking any chance.

Dr. F. W. Lamb, Cincinnati: As the essayist has described the operation much as Smith does it, the discussion should be on postoperative treatment. We do not follow the open method of treatment he has described. We do not usually look at the eye until the fifth day, unless there is some trouble, complaint of pain, etc. Then we look at the eye. As a general principle we feel we can do no good by looking at it, therefore why disturb it? If the patient is very old and feeble, we allow him to get out of bed the next day, and give him more freedom than if he is strong and robust.

Dr. H. R. Wright, Columbus, Ohio: There is no doubt that the average cataract patient in private practice is a far better risk for operation than the class of patients treated by Dr. Millette at the National Military Home.

In surgical operations age is a great factor, for with it come many affections that interfere with the nutritive processes, such as the degenerative changes of senility, involving the lymphatic and blood vessels, the heart, the kidneys, and the bladder.

Taking 10 cases of operation for cataract reported by Arnold Knapp in 1915, the average is 65.13 years. The average age of forty cases operated on by Derrick T. Vail, reported in 1916, is 64.85 years, and the last fifty cases of our practice averaged 65.2 years, thus showing in these 190 cases an average seven years less than the average of Dr. Millette's cases.

In our fifty cases, two were not in good physical condition; one had an inflammatory condition of the integument approximating the operative field, and the other was weak and despondent because of having no home and being dependent on charity. More than 75 per cent. of these cases were operated on at their homes.

In but a very few cases have we been able to follow the open method of postoperative treatment recommended by Dr. Millette, for it has always appeared to us as a needless risk. These aged cataract patients cannot be implicitly trusted. They want to feel the eye to ascertain if it is there, or raise the bandage to be sure they will see.

It has been my good fortune to be present at Dr. Millette's cataract operations and to witness his technic, which was faultless. I certainly congratulate the doctor on his absolute control of his patients and his efficient staff of assistants and nurses. It is impossible for us in private practice, not so privileged, to follow such a line of pre-operative and postoperative treatment as does Dr. Millette.

Dr. J. W. Wright, Columbus, Ohio: I want to say for myself that I began operating for cataract in 1873, and have operated for forty years, and I think that is long enough for one to use the knife. There are other dangers outside of infection. I was often obliged to operate in the country, going many miles and remaining until the third day after operating to give the first dressing, when I left it with the general practitioner to give the after treatment, and I have had few cases of infection. The greatest danger, it seemed to me, was from lenticular matter left in the eye after operation, in which case we are likely to have an iritis and occasionally a uvitis. Cortical substance does not absorb as readily in an old person as in the young, and is the cause of many failures in cataract operations. For this reason the intracapsular operation is far superior to others, when it can be accomplished with safety.

Dr. J. G. Dorsey, Wichita, Kan.: About a year ago I operated on an old man about 80 years of age for cataract. Before I left the hospital the sister came and said I would have to do something for that man; he was going crazy. He was certainly nervous. He wanted that bandage off so badly, I took it off one eye and left it on the other. The next morning I saw the bandage had been disturbed and I told him so, and he said: "Well, I took it off and had a good wash." I dressed it again and he took it off, and put it on for five days, and then it was infected and the eye was lost. About the tenth day he said he had scratched it with his finger. Old people accustomed to do what they want to will do it if they can. In speaking of ambulatory work along this line, I have done eighteen cataract operations in my office. I have explained the dangers to the patient and these cases have done as well as any cases I have done in the hospital. In many cases they have done better. I have had no accident yet. A number of times I have done it for patients who had some physical disability which made them think they could not lie down. My success is just as good as when I confine them to their back.

Dr. Walter Parker, Detroit: In an experience of 1,000 cataract extractions I have performed the intracapsular operation eighty-one times. From this experience I am satisfied that for me it should not be the routine operation, but should be performed only in very carefully selected cases.

I am very much interested in the open method of treatment. While I still think it advisable that the dressings be left on three or four days, I have noticed in cases in which the dressings have been removed earlier, either in cases of a discharge from the conjunctival sac, or in cases developing delirium, the patients have done surprisingly well. My experience in the open method treatment is, however, far too limitied to express an opinion. In regard to the reaction following cataract extraction, it is certainly surprising how many cases show reaction either on the third, eighth, or ninth day. I have never been able to determine the exact cause of the ninth day reaction, and believe there is an opportunity for some good work to be done in this group of cases.

In regard to the treatment, large doses of sodium salicylate are now given routinely in all except the specific cases, no matter at what time reaction comes on.

Question: What about the ninth day?

I think it is a low-grade iritis. Whether it is due to the remaining cortical matter, or to a mild infection at the time of the operation, I am unable to answer.

Dr. C. B. Hatch, Newark, Ohio: I did not suppose that any one today would be rash enough to follow out the method outlined by Dr. Millette. Three years ago in three consecutive cataract operations in three persons past 70, postoperative mania resulted. A little later I operated on an elderly man. The second day he became very nervous, and I allowed him the freedom of his bed. He got up and found a chair and was sitting rocking when I next saw him, and said he was going to remain up. He seemed so comfortable that I let him stay and he got along well. During the last year I operated on an old lady over 90, and she was allowed to be out of bed in a chair after twenty-four hours. Recovery was perfect. These patients had the services of a trained nurse, and the operations were done in the homes of the patients.

Dr. Hiram Woods, Baltimore: Some years ago I had ample opportunities to observe this form of dressing. When the A. M. A. met in St. Louis in 1886, the late Dr. Julian J. Chisolm obtained from a paper read by Dr. Michael his first information regarding the isinglass treatment. For several years afterward we used it at the Presbyterian Hospital, regarding the eyelid as an excellent splint, provided it could be kept permanently closed. Later, we substituted the metal shield devised by the late Dr. Murdoch. Personally, my reason for giving up the isinglass plaster was that the average hospital patient could not be made to keep his hands away from his eye. I have seen one trying a few hours after operation to test the eye just operated on. A low grade of bothersome catarrhal conjunctivitis was very common.

So far as postoperative mania is concerned, I never saw a case. Dr. Lancaster's remark is full of wisdom. I do not give these patients absolute instructions to lie still, nor do I keep them in a totally darkened room. I do try to keep the EYE QUIET. This is impossible if the fellow eye is left open. Moreover, if the patient looks down, as he generally will, the corneal wound is bound to gap.

Dr. R. S. Lamb, Washington, D.C.: I have for a number of years not closed the wounded eye. I use a moist dressing held on by adhesive straps. I allow the patient to get up. I use acetyl-salicylic and hexamethylenamin, one tablet every four hours before and after operation.

Dr. F. G. Steuber, Lima, Ohio: If the operation is "smooth," I do not look at the case for thirty-eight or forty-eight hours. If doing well, I see no reason for inspection, only to gratify our curiosity. I believe in having the patient sit up early. Prop him up the second day, and the third day let him get out of bed. As to the length of time for the bandage, that depends on the individual case. As we have all observed, the retention of cortical matter is likely to retard convalescence. I also believe in the preliminary treatment of potassium iodid, etc.

Dr. Millette (closing discussion): I am more than gratified that the paper has brought out so much discussion. Considering the class of patients in which cataracts usually occur, and the tendency to mental and genito-urinary troubles, I am firmly convinced that a practical open after-treatment, with as much freedom of action as conditions will allow, is best for the ultimate welfare of the patient. I thank you.

LANTERN SLIDE DEMONSTRATION OF ROENTGENOGRAPHIC FINDINGS OF BLIND DENTAL ABSCESSES CAUSING ACCOMMODATIVE ASTHENOPIA

AND OF AN UNUSUALLY LARGE MYXOFIBROMA REMOVED FROM THE NASAL PASSAGE

C. B. Fulkerson, M.D.
KALAMAZOO, MICH.

These case reports are not for the purpose of presenting original investigation concerning accommodative difficulties that we encounter in our refraction work. We wish merely to call your attention to some dental lesions that may cause irritated accommodation, thus perplexing the ophthalmologist and discouraging the patient. There should be a close cooperation between the dentist and the ophthalmologist. The Roentgen-ray should be resorted to more often in our cases of refraction that have symptoms which glasses should relieve but do not. Just because the teeth do not have cavities is no criterion that there is not an impacted molar or blind abscess that irritates just enough to interfere with a normal flexible accommodation.

Case 1.—Aug. 21, 1915, D. R. T., an American, male, white, aged 22, whose occupation was bookkeeper, sought relief for inflamed eyelids, increased lachrymation, and blurred vision at the near point. His general history was negative. He had never worn glasses and never had eye trouble previously. R. E. V.=7/6-4; L. E. 7/6-5. T. = N. The conjunctiva, both bulbar and palpebral, was injected. Fundus was negative. The anterior postnasal and throat examinations were negative. Homatropin was used as cycloplegic. R. E. and L. E. vision was 7/15. He accepted R. E. +0.75 sph. =+0.25 syl. ax. 75 V.=7/6 and L. E. +0.75 sph. = +0.25 cyl. ax. 105 V. 7/6. There was muscular balance at 6 m. At 30 cm. there were 5 degrees of esophoria, 12 degrees of abduction, and 10 degrees of adduction. At 30 cm. the vertical meridian was negative. The reported symptoms were relieved in sixty days. Aug. 31, 1916, he reported with irritation of both eyes. The manifest examination demonstrated no change and the physical examination was negative. Patient was referred, Aug. 31, 1916, to Dr. O. E. Lamphear for roentgenogram of teeth. Improper filling of root canals was causing some irritation, cellular infiltration and breaking-down of dental process. The tooth was treated and was still kept open to limit

irritation. His eyes improved and he was able to continue his occupation as bookkeeper. Ultimately tooth would need to be extracted, roentgenogram demonstrating improperly filled root canal in tooth that has large filling.

Roentgenogram demonstrating improperly filled root canal in tooth that has large filling.

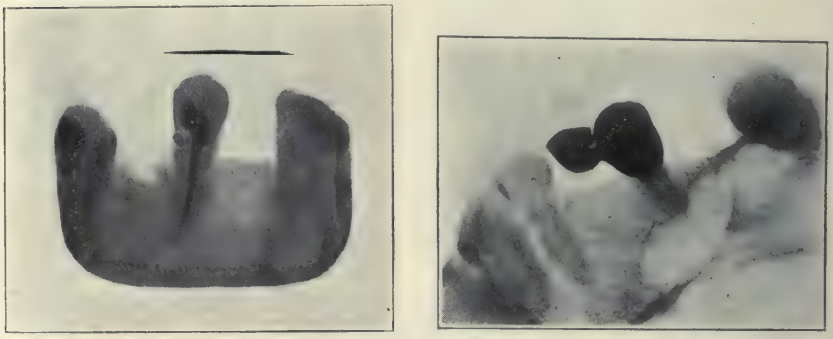
Patient returned in January, 1917, with former accommodative asthenopia. This is the photograph of tooth extracted, which the roentgenogram demonstrated as having improperly filled root canals.

Case 2.—Nov. 11, 1914, Mrs. U. L. M., aged 43, American, white, whose occupation was housewife, sought relief for pain in both eyes when reading. The patient had never been well and had had several operative procedures on pelvis and abdomen. She had always been nervous. She had worn glasses since she was 14 years old. Several refractions were made under atropin and some without. The last refraction was manifest in March, 1914. Her left eye had always given the most trouble. The conjunctiva of left eye was injected. Her vision with glasses was: R. E. 7/7, L. E. 7/7-2. Without glasses her vision was R. E. 4/12, L. E. 4/15. Ophthalmoscopic examination was negative. Under homatropin she accepted: R. E. +2.00 sph. = +0.50 cyl. ax. 75; V. 7/6; L. E. +2.25 sph. = +0.25 cyl. ax. 75; V. = 7/92. There was muscular balance at 6 m.: At 30 cm. there were 2 degrees of esophoria, 6 degrees of abduction, and 10 degrees of adduction. Fundi were negative. She accepted for reading: R. E. +3.00 sph. = +0.50 cyl. ax. 75; L. E. = 3.25 sph. = 0.25 cyl. ax. 75.

She reported in thirty days that she was relieved of some irritation. She returned April 16, 1915. The left eye was painful and irritated. Sphere was changed from +2.00 sph. to +1.75 sph. Transillumination of sinuses was negative. April 19, 1915, fields were taken and found to be normal but for some interlacing of the color fields. Jan. 1, 1916, reading point changed to +2.75 sph. both eyes.

She consulted Dr. Lamphear, dentist, Jan. 24, 1916, under whom roentgenoscopy revealed blind apical abscesses on the left upper lateral and lower molar and right upper lateral and

bicuspid. These were treated, but later extracted. There was some irritation from old fillings on the right. Patient continued treatment until July 1, when she consulted for refraction a colleague of mine, but the condition in left eye did not improve. She consulted me on Nov. 14, 1916. By manifest refraction she accepted the same correction that I prescribed on Feb. 1, 1916, R. E. +1.75 sph. =+50 cyl. ax. 75; L. E. +1.75 sph. = +25 cyl. ax. 75. For reading, R. E. +2.75 sph. =+0.50 cyl. ax. 75; L. E. +2.75 sph. =+0.25 cyl. ax. 75. Roentgenoscopy showed a healthy dental tissue and eyes were comfortable. She was much improved in health in every way.



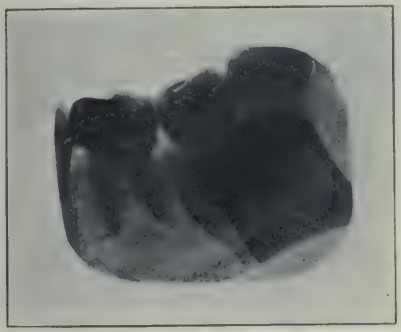
Roentgenograms of upper right and upper left showing which teeth have been extracted; also the blind abscesses that were causing the trouble. Dental process now healthy. Report on Jan. 20, 1917, stated that eyes were still comfortable with old correction.

Case 3.—March 16, 1914, Mrs. W. H. K., aged 27, an American, white, whose occupation was housewife, sought relief for headaches and blurred vision when reading. She had worn glasses from 1903 until 1913. During this period refractions were only manifest ones. She was operated on for appendicitis in 1907. Recovery was good. In 1910 had tonsillitis complicated by rheumatism. She had a first attack of suppurative otitis media in 1907. There was a slight deviation of the septum, but this did not interfere with respiration. There was some tonsil tissue present, but pus did not appear on pressure. Ocular conjunctiva was injected. Vision of both eyes was 7/6. Under homatropin cycloplegia vision: R. E. 7/9; L. E. 7/18. Accepted R. E. +0.50 cyl. ax. 105, V. = 7/6; L. E. +0.50 cyl. ax. 75, V. =7/6. She reported in thirty days she was relieved. Dec. 1, 1914, reported with eyes injected and lachrymation when reading or sewing. She accepted the same lenses she was wearing—R. E. +0.50 cyl. ax. 105; L. E. +0.50 cyl. ax. 75. She has had some earache in the right ear, but membrana tympani looks normal. The roentgenogram demonstrated an impacted molar, but dental symptoms were absent. Extraction of lower molar brought relief of symptoms of eye strain. In August, 1916, because she was nursing an infant, she did not permit the

use of homatropin for her eyes. She went to an optician for the test, but lenses did not relieve symptoms.

On Nov. 24, 1916, she consulted me again for symptoms of eye strain. Some teeth were sensitive. Refraction under homatropin demonstrated R. E. +0.62 cyl. ax. 105, L. E. +0.75 cyl. ax. 75. There was 0.5 degree of left hyperphoria; 3 degrees of esophoria at 6 m.; 2 degrees of esophoria at 30 cm.; 6 degrees of convergence and 4 degrees of divergence at 30 cm. Ophthalmoscopic findings were negative. Roentgenoscopy demonstrated blind abscess about third molar. Root canals had not been properly filled.

On relief of dental pathology, I anticipate the accommodative difficulties will disappear, because the change in lenses is too insignificant to explain all the ocular irritation.



Roentgenogram No. 1 shows pus about a molar tooth and root canals of molar tooth not properly filled. Absorption is beginning about tips of tooth.

Roentgenogram No. 2 shows former wisdom tooth extracted, yet more trouble about molar tooth that was not causing symptoms one year previous. This tooth has been extracted with relief of irritating symptoms.

Case 4.—June 23, 1916, Miss L. P., aged 30, American, white, whose occupation was office assistant. She sought relief for ocular pain and blurred vision when working at the office or reading. She had worn glasses for sixteen years. She was refracted about once a year. Sometimes refraction was manifest and other times under a cycloplegic. Her eyes had troubled her more during the last three or four months. Family history was negative. Patient had always been nervous and frequently under physician's care. Diagnosis of hysteria was made by Dr. R. G. Cook. For three months had had neuralgia, pain about head and face. Dental work had just been completed. (I might say that the dental work was done by a dentist who belittles roentgenoscopy in dentistry.) Nose and throat examination was negative. Vision R. E. 7/7; L. E. 7/9+2. Under homatropin cycloplegic vision was: R. E. = 4/45; L. E. 2/22. She accepted R. E. +3.50 sph. = +0.50 cyl. ax. 45, V. = 7/6-1; L. E. +4.50 sph. = +1.00 cyl. ax. 150, V. = 7/7+2. There were 4 degrees of esophoria at 6 m; 4 degrees of esophoria at 30 cm.; 3 degrees

of convergence at 30 cm., and 6 degrees of divergence at 30 cm. Postcycloplegic test July 7, 1916, gave: R. E. +2.25 sph.=+0.50 cyl. ax. 45; L. E. +2.75 sph. =+1.00 cyl. ax. 150. Reported Sept. 1, 1916, as still having trouble. Roentgenoscopic findings were as follows: Improper root filling and irritation. Dental treatment continued and eye condition improved, but was aggravated as soon as caps were replaced. It improved as long as tooth drained.

We believe that prophylactic dental therapeutics will not produce definite results in many of these cases. Extraction is the proper treatment.

Roentgenogram of Case 4.

Case 5.—Aug. 5, 1916, Mrs. C. O. K., aged 30, American, white, whose occupation was housewife, sought relief for headaches and pain in eyes after reading. The patient has a small goiter, some obscure nervous symptoms, and a pulse of 95. The patient has worn glasses since 1906. Refraction was taken under a cycloplegic. Last refraction was one year ago. The nose was negative. Transillumination of sinuses was negative. Throat was negative. Conjunctiva was generally injected. Vision R. E. and L. E. 7/7-4. Under homatropin cycloplegia she accepted: R. E. +0.50 sph. =+0.25 cyl. ax. 105; L. E. +0.50 sph. =+0.25 cyl. ax. 105. There was 1 degree of exophoria at 6 m.; 8 degrees of exophoria at 30 cm.; 1 degree of abduction and 5 degrees of adduction at 30 cm. There were 2 degrees of exophoria in accommodation. Ophthalmoscopic findings were negative, but there was a suspicion that retina was more red than normal. She reported in thirty days that condition was improved.

May 1, 1916, with return of symptoms, she reported again, stating she had been under treatment with Dr. Alice B. Ellsworth for hyperthyroidism, periods of nervous irritation, and cardiac arrhythmia. Manifest examination changed R. E. lens to +0.25 sph. =+0.25 cyl. ax. 60; L. E. lens to +0.12 sph. =+0.37 cyl. ax. 105. Also referred her to Dr. O. E. Lamphear, under whom roentgenoscopy showed an impacted lower left. Extraction gave relief of symptoms. I was in doubt whether to attribute accommodative symptoms to the teeth or to hyperthyroidism.

During September, 1916, her old symptoms returned. She did not consult me, but went to Dr. Lamphear, who took a roentgenogram of the entire upper and lower maxillary processes, finding in the upper another impacted molar, and in the lower right tooth there were unfilled root canals and one dead tooth with canal not filled. No doubt some of this pathology existed in May. Fluoroscopic examinations are not as satisfactory as plates.

Roentgenogram.—Impacted molar causing trouble in May, 1916. Its extraction brought prompt relief.

Roentgenogram was taken in October, 1916, when old symptoms of irritation had returned. When extracted complete relief was afforded.

Case 6.—Nov. 14, 1916, Mrs. M. E. E., American, white, aged 30, whose occupation was housewife, sought relief for blurred vision. August, 1916, she had a spinal operation. She was quite nervous. She began to wear glasses in 1901. She was refracted under a cycloplegic. At that time she had symptoms of blurred vision and nausea. She did not have glasses changed again until August, 1916, which was a manifest refraction. She was wearing R. E. +1.25 sph. =+0.25 cyl. ax. 90, and L. E. +1.50 sph. Vision with glasses R. E. 4/9+4; L. E. 4/22. Vision without glasses R. E. 4/30; L. E. 4/45. Septum was irregular and deviated to the left. There was evidence of partial turbinectomy on left side. Tonsils showed crypts filled with pus. Pupils reacted to light and accommodation. T=N. The optic disk was larger than normal in the vertical meridian; media was clear; there was a choroidal ring around the disk of both eyes. Under atropin vision was: 7/21 R. E., 7/30—1 L. E. Nov. 6, 1916, she accepted R. E. +100 sph. =+0.50 cyl. ax. 90, V. =7/7+3; L. E. +1.25 sph. =+0.50 cyl. ax. 90, V. =7/7+1. Muscular balance was at 6 m. There was 6 degrees of adduction and 4 degrees of abduction at 30 cm. Retina had a fuzzy appearance at this examination.

Ten days later patient reported with irritation about eyes. Had a tooth which was sensitive. Roentgenogram was taken of whole dental tissue and a small root was found on one side and a small opening into the antrum. This case is too recent to

determine outcome. Roentgenoscopy in this case has isolated sufficient cause for irritation. A definite therapeutic procedure can be outlined for irritated accommodative symptoms which might have been attributed to a general unstable nervous system, or irritation coming from a remote area in the spine.

Roentgenogram of Case 6 shows an old opening into antrum and a small root, both of which were causing some irritation.

Case 7.—Sept. 6, 1916, Miss F. H., American, white, whose occupation was housewife, sought relief for nasal obstruction. Lump high up in the throat and back of the palate. Seven or eight years ago she had beginning nasal obstruction. Had polypi removed by Dr. LaHeuis in 1906. Since 1910, condition had been gradually getting worse. There was a deviated septum to the left high up. The right passage was occluded by a large tumor which resembled a polyp.

Sept. 9, 1916, under local anesthesia, removed with snare the anterior part of the growth, which permitted one to see where the remainder was attached to the lateral wall of the chamber between the middle and inferior turbinate. This was cut off with a pair of turbinate scissors. The growth then dropped back into the postnasal space and was removed through the mouth.

Examination showed the septum pushed to the left, so that the middle turbinate was pushed outward and was more or less degenerated. The ethmoidal cells on the right side were soft and filled with pus. The middle turbinate was removed and the ethmoidal cells exenterated.

The pathological findings were as follows: The nasal polyp proved to be a myxofibroma. The central portion was composed of delicate fibrils showing a moderate number of nuclei, with spaces between the fibrils occasionally containing a homogeneous pale substance. The peripheral portions showed a focal proliferation of the cells of the mucosa; nowhere did the cells break through the basement membrane. The underlying tissues were edematous, and showed a rather heavy infiltration with compound granular cells, lymphocytes, and occasional plasma cells, with a few polynuclear cells. In some portions of the section

the infiltration was rather marked. Many blood-vessels throughout the entire section were greatly thickened, sometimes manifesting an obliterative arteritis. They were generally infiltrated with lymphocytes and compound granular cells. The endothelial lining showed a marked proliferation in many of them. The vascular condition and peripheral infiltration was rather suggestive of a possible syphilitic etiology.

Photograph of myxofibroma.—Dec. 30, 1917, under local anesthesia a submucous resection was done. Linear incision was made on left side of septum. The central part of the septum was thin and atrophied by pressure of the myxofibroma which was removed at the former procedure.

DISCUSSION

Dr. Edward Stieren, Pittsburgh, Pa.:—There is no doubt that the rôle the gums and teeth play in diseases of almost any structure of the eye is of prime importance. In fact, I do not believe we exaggerate if we declare them to be of the highest importance. As the essayist has said, the difficulty in achieving results in this class of cases is to secure adequate team work with the dentists. Unfortunately, dentists, being taught to conserve the teeth as much as possible, are loath to believe that a tooth may have been properly treated and yet remain a source of danger to the owner. Time and time again these cases come up, and it is only by the most careful roentgenoscopy applied to the patient's teeth that these blind abscesses are discovered, and it is strange that the dentist should take this wrong attitude. I remember that, three or four years ago at a meeting of the college of Physicians of Pittsburgh, at which Dr. Thayer of Johns Hopkins was the honor guest, he chose this same subject for his address. At this meeting we had the dean of the dental college present and some of the faculty. They were enthusiastic over the result to be achieved in cooperation with the medical men, but, even to this day, we find the majority of dentists are not alive to the importance of their work in aiding the medical man. There is no doubt that almost any disease of the eye can be caused by absorption of infectious material from the teeth and gums. The essayist has reported a series of cases of lost accommodation.

I have seen but one or two. The deep inflammatory conditions which usually baffled us are now cleared up by our increased knowledge of the important rôle that the teeth play, and, as time goes on, I predict that cases usually considered to be of rheumatic, tuberculous and syphilitic origin, will fall into the dental class.

Dr. T. E. Carmody, Denver:—I would like to say just one word. The essayist spoke of not showing much in his roentgenograms. I have found, in a great many cases, the roentgenogram does not show anything. There may be a great deal of destruction around the tooth, and yet the roentgenogram does not show anything. One root canal, he said, was almost clear over the end. I have found in those cases in which they are filled only to within one-sixth or one-eighth of an inch from the end, the serum collects and we have frequently infection of the face. I have found a great many of these cases produce throat symptoms, which clear up on removal of the tooth.

Dr. Fulkerson, Kalamazoo, Mich. (closing discussion):—The smaller the number of teeth included in the roentgenogram the better the result will be. Now they use from three to six exposures, whereas at the beginning one exposure or two were thought sufficient.

TRANSACTIONS

OF THE

TWENTY-FIRST ANNUAL MEETING

OF THE

American Academy of Ophthalmology and Oto-Laryngology

OTO-LARYNGOLOGICAL DIVISION

HELD AT
MEMPHIS, TENN.,
DECEMBER 11, 12, 13, 1916

SHOULD THE OTOLARYNGOLOGIST ENLARGE HIS FIELD DIAGNOSTICALLY, TECHNICALLY, OR BOTH?

VICE PRESIDENT'S ADDRESS

T. E. Carmody, M.D., D.D.S., D.D.Sc., F.A.C.S.
DENVER, COLO.

Within the last decade we have seen many changes in our specialty, most of which have tended toward a contraction of the field, with the result that better work has been performed. However, we should not allow ourselves to seek contraction at the expense of efficiency, which may happen if we carry this idea too far.

It is a question whether we should always enlarge our field technically. Especially is this true of many operations that we may consider borderline on account of experience of the individual operator. Should we allow this to be true?

Many otologists perform operations on the organ in their field, and yet do not consider themselves, or any other otologist, competent to remove the internal jugular vein if thrombosed, but will call in a general surgeon. While this may be right in some individual cases, it allows the impression to stand with the general profession that we are not competent to deal with serious cases, and, judging from criticisms from surgeons called in on these cases, we are not considered surgeons.

On the other hand, the general surgeon believes he is competent to operate in any surgical case and is jealous of any encroachment, so the criticism, at times, may be given in that spirit.

The following surgical conditions should be in the hands of the otolaryngologist, or he should, at least, be competent to deal with them, although frequently given over by him to others:

(1) Resection of internal jugular, in cases of infection from mastoid;

(2) Ligature of external, internal, or common carotid, in case of injury during operation on the throat, or in case of infection of the glands, or a cellulitis of the neck;

(3) Removal of infected lymph glands; operations on the thyroid gland.

(4) Operations on the palate.

Little need be said about operations in brain cases, as we have taken this field, except for tumors and fractures of the skull itself.

Having with success turned our attention to the brain, we have also, if possible, made a much more successful entry into the chest through the trachea and bronchi, and even through the chest into the abdomen, by way of the esophagus to the stomach. The success in the former can be judged by referring to the writings of Day, Kopetsky, Rutin, Beck, and others. While the work along the latter line needs very little comment, we may speak of the work of Killian, Jackson, Brueing, Johnson, and Lynch as the foremost. While the achievements in these spheres of action have been epoch-making, have we not gone far afield and overlooked something nearer? May it not be said that we have neglected our opportunities?

Would it not be well for us to think long on the methods of voice production and control, and less of the removal of tissues in which there is a question of their pathology? Should we not pay more attention to the defects of speech, resulting from congenital defects of nose, mouth, pharynx, and larynx, as well as to those unfortunates who, having known the pleasure of normal speech, have lost it through loss of the speech producing mechanisms, or the sound perceiving apparatus? We pay too little attention to these, I am afraid, because they do not bring returns in this age of keen competition and high prices. All the more credit is due the work of Makuen, Goldstein, and their few associates in this field. How much do we study the sound producing apparatus? What do most of us really know as to the association of the different parts of the machine?

Is it to be developed as a part of our specialty, or should we assign the work to someone else? Should we try to correct the defects, or say it is lack of co-ordination and leave it to the neurologist? Should we master it in all its details in order to help others, or should we leave it until it is forced upon us by the rest of the profession?

In speaking of the organs through whose action or passive resistance the voice is produced, the mouth is next to the larynx in importance, more, it is true, as a modifier of tones, but nevertheless necessary to speech. The question naturally arises,

what do we know of it and what are we taught about it, either in our medical schools proper or our postgraduate courses? The answer requires but one word—nothing. Why has this most important cavity been ignored by the medical profession? Why is it, as Upson expresses it, "An Ishmael not to be admitted to its pathologic birthright"? It is mainly because our forefathers in medicine did not see the necessity of keeping dentistry under the wing of medicine, but allowed it to split off with the result of almost complete alienation, so far as the dentist is concerned. Apparently "Mother Medicine" did not know her child.

Medical literature, journal articles as well as textbooks present a pathetic figure in attempts to treat of technical conditions in the mouth and of the teeth in general. While we believe we are fully awakened at this time, we have yet far to go.

We have apparently awakened to only one side of the relation of the mouth to the organism in general, and that is the side that is uppermost in our minds today, because of our interest in the science of bacteriology—infection. While not denying the good which may come of this, we may go to extremes, because of our very lack of breadth, which is usually true of innovations, whether they have to do with pathologic conditions per se, or the conduct of specific operations.

We must, furthermore, not be carried away with the idea that all pathologic conditions about the mouth can be immediately and unfailingly diagnosed by roentgenoscopy and take these findings against clinical evidence. Unless we give full weight to both we shall make many mistakes. On the other hand, we must not decide rashly that all infection originates in some pathological condition of the mouth.

While we have long been interested in the pathologic rôle of the tonsil, we have looked through and beyond the cavity which is a very incubator of pathological conditions, and yet have not recognized them. It may be there are many exceptions, but I venture to say that, until the last two years, the great majority of us did not know whether our patient's mouth contained teeth, with the possible exception of the six anteriors above and below, and whether they were alive, dead, or the crowns of even these were natural or artificial.

This being the case, how much more likely is the so-called blind abscess to be overlooked, to say nothing of pyorrhea pockets that might have destroyed much of the process, producing in either case an overwhelming toxemia, or very

pronounced reflex disturbance, or both. Furthermore, we should ever discover the cases in which some of the teeth had failed to erupt, the pernicious influence of which is well known and which are easily found by the aid of the roentgenogram.

It is my hope that more attention will be paid to these subjects in our medical schools and especially in our postgraduate courses. We shall all learn that all parts of the head are so closely related that no part of it should be overlooked, or its pathologic significance forgotten, when dealing with some other parts of the body.

Learn the normal picture of the mouth, face, and neck first, and then we shall know the variations from the normal, whether we depend on our eye alone, or resort to other aids in diagnosis.

IMPORTANCE OF THE INTERNAL SECRETIONS IN EAR, NOSE, AND THROAT AFFECTIONS WITH SPECIAL REFERENCE TO THE HYPOPHYSIS

HARRY L. POLLOCK, M.D.
CHICAGO

As you have learned from Dr. Beck's paper, the great problem which confronts us in our work with the ductless glands is the difficulty, or rather the inability, by any laboratory examination to determine whether we are dealing with a hypersecretion or a hyposecretion of one or more of these glands.

It is only by clinical symptoms which present themselves after a great change in the glandular secretion has taken place that we are able to judge approximately what gland or glands are affected. Not until the physiologic chemists can definitely tell us by examination of the blood and the various excretions or secretions, whether there is a deficiency or an overproduction of the secretions, shall we be able to put our treatment of these glandular disturbances on a rational basis. However, the knowledge of these mysterious structures has been gained experimentally in a twofold manner: (1) By an overfeeding or implantation of the glands themselves in animals and noting the difference between the animals so fed and the corresponding controls; or (2) By removing, or partially removing, the glands by surgical methods and then observing the results obtained thereby. In the beginning, this second method was very difficult to apply, owing to the fact that it was almost impossible to reach or remove these glands without the animals dying from the immediate effect of the operation (usually from shock), before the symptoms arising from the removal could be studied. The thyroid and parathyroid could be removed very easily and safely, and the effects studied, but when the removal of the hypophysis, the thymus, the pineal gland, or the suprarenals was attempted, the animals usually died so quickly that results from the effects of the surgical interference could not be obtained. For instance, Restelli, who in 1845 first attempted to study the effect of the removal of the thymus, lost ninety-two of ninety-eight animals operated on, and the remaining six lived but such

a short time that no conclusive results could be obtained. After aseptic surgery was introduced and surgical technic improved, the animals so operated on lived until they either died from the effects of the loss of the secretion or were killed in order to obtain and study the pathologic conditions and changes brought about by the lack of the necessary ductless gland secretion. During the past decade so much has been written on these various glands that an attempt to review the entire literature would occupy too much time, and it is not in the province of my paper to do so, but I will attempt in a few words to give you the status as it is now accepted by most of the authorities, with, however, a more detailed study of the hypophysis. As the thyroid is the most accessible, and the disturbance either of a hypersecretion or hyposecretion, is known to all of you, I will not discuss this gland, except to state that a hypersecretion results in the condition described first by Basedow and known as exophthalmic goiter, which has been described to you by Dr. Beck. A hyposecretion in children results in cretinism and in adults in myxedema. All authorities agree that to be in perfect health all the ductless glands must work in harmony, and when one or the other is working excessively or not secreting sufficiently, the glands are thrown out of harmony, i. e., they must undertake to supply the deficiency so caused, or an antagonistic gland will secrete excessively, as the inhibitory influence of the affected gland has been removed. We know that the suprarenal as well as the hypophysis acts as a check on the thyroids, and vice versa, and that often when the suprarenal is destroyed, or is secreting too little, the hypophysis will hypertrophy and attempt to replace in the economy that which is lacking by reason of the insufficiency of the suprarenal. To what cause this disharmony is due is still an open question; yet when we at times see such brilliant results obtained by the removal of some focal infection, we cannot but believe that these focal points play a great rôle in the causation of the disharmony and must always be taken into consideration when we study these cases. As Dr. Beck has already explained our views on this subject and given illustrations thereof, I will only emphasize this one point—*search carefully for various focal points of infection in any change in the ductless glandular secretion.*

The Thymus.—This organ was formerly supposed to be an embryonic one and after birth was expected to gradually retrograde. Karl Basch, in 1902, in experimenting on dogs, which

he found were best for his work on the thymus, discovered that this gland continued to develop for at least four weeks after birth, and then retrograded very rapidly. In the human the retrograde action occurs until puberty when the gland is supposed to have disappeared.

If the animals were operated on after the fourth week, few, if any, changes occurred in the subsequent growth, but if the gland was removed previous to the fourth week, marked changes in the development took place. The change took place mostly in the bony structures. The bones became soft and weak and the animals could not stand. There was a loss of the calcium salts, and when a fracture was produced it healed very slowly. The union was usually a fibrous one, and it took months before there was a gradual bony union.

This condition corresponded strikingly with the rickets of children. The extirpation of the thymus also heightened the irritability of the nervous system. The thymus also has a distinct relation to the sexual apparatus as shown experimentally. A persistent thymus shows an undevelopment of the genitalia, a castration showing a persistent increased size of the thymus. On the other hand, thymus extract injected intravenously manifested itself as a depressant to the heart, caused a slowing of the pulse, and a fall of blood pressure, until death resulted. Postmortem findings always showed a clot in right ventricle extending into the pulmonary vein which was reported to be the cause of the death. If, however, medicine (hirudin) was first injected which tended to prevent clotting of the blood, a similar lethal dose of thymus could be injected without any symptoms whatever. Sudden deaths from ether in status lymphaticus or persistent thymus is caused by this blood clotting in the right ventricle, and if anti-clotting medicine (hirudin, for example) be first employed, this danger can be obviated.

The Suprarenals.—These glands have been studied since Addison, in 1855, first described the disease which bears his name. He described the etiology as due to the destruction of the suprarenal glands. Many animal experiments have been made by the total removal of both the cortical and medullary portions, but it is impossible to remove either portion separately, as they are so intimately connected. Histology proves to us that the main secretion arises from the medulla and from this the extract is obtained. The symptoms from the removal of this gland are a progressive muscular weakening and a malaise. There is

a cordiovascular asthenia shown by a gradual weakening of the heart action, and very often, but not always, a pigmentation of the mucous membrane and skin. The study of the effect of adrenalin on the various organs, excretions, blood, and metabolism is so complex that I will not attempt to describe this in detail, but will consider only those things which are of interest to us as otolaryngologists.

The first effect of an injection of adrenalin is the immediate rise of the blood pressure and this is brought about by a constrictor action on the wall of the vessels themselves, and not by a stimulation of the heart. This is so thoroughly proven experimentally that the suprarenals are called the glands of the blood vessels. The action on the vessels is of short duration, and very often succeeding the constriction there is a dilatation shown by a fall in the blood pressure. Long continuation of these adrenalin injections causes a necrosis of the middle layer of the arteries and a deposit of lime salts therein, and arteriosclerosis is now thought to be due to an effect of the overproduction of the suprarenals, i. e., a slow poisoning by the gland secretion. Of greater importance to us, as we shall see, is its action on the bones.

Bossi found experimentally that in sheep in which one gland was removed there resulted in a few days symptoms of osteomalacia, viz., phosphaturia and osteoporosis, which was proven by roentgenograms. He utilized this discovery practically, i. e., he treated cases of osteomalacia with adrenalin injections and found that his patients recovered completely, and this is now accepted as the rational treatment of this disease.

By the courtesy of Dr. Carl Beck, I have some lantern slides of a case of osteomalacia which will be shown, and in which the patient fully recovered after hypodermic injections of adrenalin. What value has this knowledge to us as otolaryngologists? We know histologically that in otosclerosis, in the early stage, there is an osteoporosis of the bony labyrinth, the same histopathologic condition as in osteomalacia. If adrenalin can cure these latter conditions, why can it not do the same in otosclerosis? It is with these facts as a basis that we are employing this treatment in our cases of otosclerosis as well as in the other conditions outlined by Dr. Beck.

In a paper read last winter before the Clinical Society of the North Chicago Hospital, I drew attention to the pathologic action of the various ductless glands on the autonomic nerve

system, and explained the theory why an irritation of this system would cause an attack of asthma, and why an injection of adrenalin would abort the same. I am still doing work along this line, but as it is at present only a theory, will not go into details at this time.

The Hypophysis.—As has been mentioned before, this is the gland that we desire to discuss more in detail, as you will see that in its pathologic changes and the various operations to which it is now subjected, it is in closer contact with the rhinologist than are the other ductless glands.

Anatomy.—Situated in the sella turcica, it is composed of three parts. The anterior or glandular, the posterior or nervous, and the pars intermedia, composed partly of both lobes. One would suppose that the anterior glandular lobe would be the portion which produced the main secretion of the gland, yet such is not the case, as it has been proven experimentally that the posterior lobe furnishes the principal physiologico-therapeutic extracts. The action of the extract is very similar to adrenalin, but differs in some of its actions on the various organs of the body, in which they act antagonistically. For instance, the action of both are increased as mydriatics when used jointly. On the muscles of the intestines, the adrenalin contracts, the hypophysis relaxes, and when used together the combination has a neutral effect.

The action of adrenalin on the circulation is far stronger than that of hypophysis, yet the latter acts longer and can be used indefinitely, while there is great danger in continuous use of adrenalin. Not until recent years were experimental operations for the removal of the gland successful. It was presumed that following the total removal of the glands, the animals died from the absence of the necessary secretion; but later it was found that such operations could be done and the animal survive. Although we know that the active physiologico-therapeutic extract lies in the posterior lobe, this portion can be removed with impunity and the animals show but little change, but if the complete anterior lobe, or the greater portion of it, is removed, the animals immediately remain stationary in their development; there is a great deposit of fat and a nondevelopment or a retrogression of the genitalia.

Clinically, and that is what interests us mostly, we are able to distinguish two distinct conditions of the hypophysis, one a hypersecretion and the other a hyposecretion.

P. Marie was the first to connect the condition of acromegalia with a hypophyseal tumor, and he thought that the disease was the result of a hyposecretion, as the gland was destroyed by a tumor formation, such as sarcoma, carcinoma, adenoma, glioma, etc. This was soon shown to be incorrect, as many cases of acromegalia showed an apparent normal condition of the gland on postmortem examination, and by roentgenoscopy the sella was found enlarged. It was soon found that this disease was caused by a hypersecretion, but not necessarily an enlargement of the gland. Likewise we find a hypersecretion of the thyroid in exophthalmic goiter, with usually a very small thyroid gland. Several cases in which there was an improvement in the condition after a removal or partial removal of a tumor of the anterior lobe added further weight to the proof.

Hochenegg, in 1908, cited the first case of acromegalia in which the hypertrophy was removed by the nasal route, and in which there was a diminution in size of hands and feet noticeable within eight days after operation. Within three months there was a complete recovery.

Since then others have cited similar results. We have recently seen a typical case of acromegalia of long standing, in which the roentgenogram showed an enlargement of the sella and a disappearance of the clinoid processes. The patient consulted us on account of his laryngeal condition. His voice became husky, and at times he had impairment of breathing. Upon examination of the larynx by suspension laryngoscopy, we found an immense hypertrophy of the mucous membrane of the entire larynx, so that it hung in folds. The microscopic section of a removed portion showed hyperplasia of the mucous membrane and the underlying tissues.

This case has already been reported in detail by Dr. Beck before the Chicago Ophthalmological Society, and will appear in the current number of the *Annals of Ophthalmolgy;* but I simply desire to call your attention to this unusual condition of the larynx, which I am unable to find mentioned anywhere in the literature. This case proved to be a mixed one, i. e., while a true case of acromegalia, he had had this thyroid removed many years before and subsequently developed a lingual thyroid which a second surgeon removed a short time previously. He then developed symptoms of myxedema.

I now desire to call your attention to another condition, the etiology of which is supposed to lie in some disturbance of the

hypophysis, but of which we have no definite proof, viz., diabetes insipidus. Many authors have shown that a disturbance of the pars intermedia, either by injury, pressure, or disease, will in a large percentage of cases be followed by diabetes insipidus.

The condition of hyposecretion was first described by Froehlich in 1901, which he called dystrophia adiposa genitalis. It is now universally known as Froehlich's syndrome. It occurs mostly in young male adults; is characterized by a deposit of fat over the entire body, but especially in the breasts and over the pubes. An atrophy of the genitalia and a corresponding loss of its function and a falling out of the pubic and axillary hair also occur. Naturally, with this comes the eye symptoms of any hypophyseal tumor, viz., a bitemporal hemianopia, resulting in complete blindness if the condition is not corrected. One of the noticeable co-existing conditions is the great tolerance to sugar. These cases can ingest an immense amount of sugar without any showing in the urine.

In this conncetion, I desire to report a case of this kind which I have just operated on by the method first suggested by my associate, Dr. J. C. Beck, and presented before this Society at the Chattanooga meeting in 1913. This consists of attacking the sella turcica by the antropostethmo-sphenoidal route.

Case 1.—Mr. O., aged 32 years, was referred to me by an otolaryngologist of Vancouver, B. C., on Nov. 7, 1916, and gave the following history: He had been married seven years, but had no children.

For the previous six months patient had been gradually growing blind. With right eye was unable to tell light from dark and in the left eye was partially blind. Condition came on gradually, blindness starting from outside of eye and going inwards.

Weight of patient had increased markedly during the previous two years, especially around the abdomen. Never had had heavy beard or mustache; had not at this time. Skin was not dry, only scalp was quite dry and hair falling out. Appetite was enormous. Bowels were regular. Urinated often and more than the usual amount.

Patient did not complain of headaches, nor did he perspire. Finger nails were sound. Could not breathe through the nose very well.

Nasal Examination.—There was a general turgescence through nose. Septum was fairly straight. Ridge was low down on left side. Inferior turbinate was turgescent.

Teeth were in repair and excellent condition. Pharynx was normal. Small tonsils were degenerated.

Ears.—Had little difficulty in hearing in right ear. Examination showed slight amount of cerumen present.

Left ear was in the same condition.

Advise roentgenoscopy and a thorough blood and eye examination.

The roentgenogram showed a flattening out of the sella and a disappearance of the postclinoid process.

The blood examination was as follows: Red, 4,800,000; white, 8,000; differential, small lymphocytes, 34 per cent.; large mononuclears, 5 per cent.; polymorphonuclears, 61 per cent.

The amount of a twenty-four hours' urine specimen was normal; 1,800 c.c. and no pathologic findings.

I referred the patient to Dr. Harry Gradle for eye examination, and he reported as follows:

Right Eye.—Complete blindness.

Left Eye.—The temporal half was gone; on the nasal half the field was constricted for all colors.

Ophthalmoscopically.—Right eye, fundus was normal, except for a slight dilatation of the veins.

Left Eye.—There was advancing secondary atrophy of the optic nerve. It was impossible to measure blind spot, as this is included in the scotoma.

Patient was given 250 grams of dextrose and urine was found to be free. Two days later 350 grams of dextrose was given and again urine was found to be free, showing an increased tolerance for sugar.

The diagnosis of a hypophyseal tumor having been established, an operation was advised. As before stated, I decided that the best and safest operation was the one suggested by Dr. J. C. Beck. This consisted in attacking the sella by the antro-postethmo-sphenoidal route, and has the advantage over other intranasal methods, in that the space through which we work is much larger and at least one to one and one-fourth inches closer to the sella than any other intranasal method.

Therefore, on Nov. 12, 1916, I did a preliminary left-sided middle turbinectomy and a thorough ethmoid exenteration. I chose the left side, as the tumor was pressing more on the right side, as shown by the complete right-sided blindness.

On Nov. 21, 1916, I completed the operation as follows:

Exposing the anterior wall of the antrum through the canine fossa in the usual manner, I removed the wall with the electric burr. Then with a curette I broke through the postethmoidal wall at the junction of the sphenoid, entering the sphenoidal cavity. After breaking down the septum dividing the two sphenoids, I attacked the posterior superior portion of the sphenoid with a special long electric burr and took away the roof, thus exposing the hypophyseal region. A soft tumor mass was felt, but I decided not to attempt to remove it at this time, depending on the effects of the decompression, and, if necessary, at a later time to remove it completely.

Patient made an uneventful recovery from the operation, but too short a space of time has elapsed to expect any great amount of improvement in his symptoms.

This is the third case of hypophyseal tumor which we have operated on by this method, the former two having already been reported by Dr. Beck; the first case, a cyst of hypophysis. Now, after two and one-half years, patient has had a great improvement in all his symptoms, and is working every day, something impossible for months prior to the operation.

The second case recovered from the operation, a tumor of the hypophysis (sarcoma) having been found, but succumbed to the disease some months after leaving our care.

RELATION OF THE GLANDS OF INTERNAL SECRETION TO OTOLARYNGOLOGY.

JOSEPH C. BECK, M.D.
CHICAGO

Our aim today shall be to bring before you this complex subject in the form of a plea for the recognition of the importance of these glands in otolaryngologic practice, in the broadest sense. Not only do we ask you to recognize them as important, but we wish to present our studies and investigations in such a manner as to enable you to cooperate in order that we may come to some definite conclusions.

The subject is a very large one, and consequently, in order to cover it, we intend to bring out only the vital and practical points, leaving the details for some future time. We will attempt, however, to cover the entire subject in a general way, and expect from time to time to take up the affections of individual glands as related to the ear, nose, and throat. Today we wish to discuss only the hypophysis in detail.

I will first describe our own studies on ductless glands, and subsequently Dr. Pollock will take up a consideration of the literature of the subject. The principal conditions that we shall consider are:

(1) Hyperadrenalism and hypoadrenalism.
(2) Hyperthyroidism and hypothyroidism.
(3) Hyperthymism and hypothymism.
(4) Hyperpituitarism and hypopituitarism.

From this outline it will be noted that the four great glands which we are considering as causing disturbances are the thyroid, thymus, hypophysis and the adrenals.

The various other structures that are considered as ductless or endocrinal glands, or partly so, such as the pineal, parathyroids, ovaries, testicles, liver, etc., we will not take into consideration at this time.

The various otolaryngological subjects that we will consider are: (1) Atrophic rhinitis. (2) Hyperplastic or nonsuppurative sinuitis, in which we have (a) hay-fever, (b) hyperesthetic rhinitis with or without hydrorrhea, (c) asthma. (3) Septal deflection and excrescences. (4) Otosclerosis.

(5) Exophthalmic goiter. (6) Myxedema. (7) Status thymicus and lymphaticus. (8) Epilepsy. (9) Acromegalia. (10) Froehlich's syndrome.

As stated before, in the study of these glands it is not our purpose to take up any other part of these diseases except that which concerns the otolaryngologist, and then only the essential points. That these glands of internal secretion play an important part in the human economy there can be no doubt. When, for instance, the thyroid is secreting excessively, there is development of rapid pulse, marked exhaustion, etc., and should one administer a dose of thyroid extract, one would see how very much these symptoms are aggravated. A dose of pituitrin will unquestionably cause marked contraction of nonstriped muscle fibers, particularly of the uterus. Again, a deficiency of thyroid secretion will be very quickly shown by symptoms of myxedema, and when thyroid extract is given, these symptoms quickly disappear. Many examples could be described of the specific action of these glands, but that is scarcely within the province of my part of the program and will be taken up by Dr. Pollock. There is still a very important portion of the work unfinished, and that is the determination, either by chemical or other tests of the blood or other secretions, as to whether an individual has an excess or deficiency of any one or more of the glandular secretions.

The pathologic changes supposedly due to alterations in the internal secretions are also conclusive evidence of the relationship, and that has been the basis of most of our work. I have special reference to the bony changes found in the nasal and aural structures. At the conclusion of Dr. Pollock's paper I will demonstrate some of the conditions.

Two glands are known to have a specific influence on bone growth, and they are the hypophysis and adrenals. The thymus, too, has some influence on the nutrition of the bone. From our histopathologic findings of the turbinates, septum, and internal ear, compared with the histopathology of the bones of the rest of the skeleton in conditions in which the glands of internal secretions are disturbed, as, for instance, in osteomalacia, acromegalia, etc., we are convinced that there is an analogy that is worth following up. In other words, the pathologic changes in the bony capsule of the labyrinth in otosclerosis are analogous to the bony changes in osteomalacia, and to a certain degree in the turbinated structure in the diseases mentioned above, as well

as to the bony changes of the septum at the point of deflection and ridge formation.

What does that all mean? Simply that we believe that in conditions such as atrophic rhinitis, hyperplastic ethmoiditis (whether we have hay-fever, hyperesthetic rhinitis, or asthma) otosclerosis and septum deviations, the same etiologic factor plays the part and the treatment should be the same.

(1) *Atrophic Rhinitis.*—Several years ago I observed that this condition occurred mostly in girls or women, and that many of these patients had small thyroids. I began empirically to feed them on thyroid extract, and they showed some improvement. I then referred every case of atrophic rhinitis (genuine ozena) to Dr. Pollock to follow out this line of treatment, and this he has done, employing the thyroid, thymus, adrenal, and hypophysis glands in a systematic manner, that is to say, not all at one time in any sort of a dose, but in particular combinations and dosages. This he reported in 1912. We have not ignored other methods of treatment, particularly the bacterial or vaccine, but have found no specific benefit from that treatment alone. However, in combination with organotherapy there appears to be better results obtained. One treatment that is beneficial in atrophic rhinitis which I may be pardoned for mentioning here is the surgical. I have recently employed a method to supplant the intraseptal injections of paraffin, which I believe to be original, although Dr. Iglauer reported some transplanting experiments a few years ago. We simply do a septal resection of cartilage and bone through a small slit. Then through an ear speculum introduced into this slit we pack small pieces of fascia (no fat).

Our theory as to the etiology of atrophic rhinitis, which applies also to the other conditions about to be described, is about as follows: Primarily, there is a chronic focus of infection. This may be intestinal, tonsillar, dental, or in the sinuses, etc. This focal infection produces marked changes in some of the glands of internal secretion, thus disturbing their entire harmonic action. In consequence of this disturbance, there result certain pathologic changes, particularly in the bony frame work—in this case, in the turbinates. It is a most rarefying process. The mucous membrane covering these bones undergoes a secondary degenerative change, and in atrophic rhinitis a metaplasia of the epithelium. Finally, there develops a low-grade infection of a great variety of organisms, among which there is the so-called specific fetid bacillus of ozena.

We therefore give these patients, either by mouth or hypodermically, glandular extracts alone or in combination, for a period of from one to six months, and after a lapse of time, say from one to three months, we repeat this treatment. It is noteworthy that at times there does not appear to be any effect from the treatment, while at another time the same treatment is followed by striking results. Why that is so we have not been able to ascertain, and cannot even suggest a theory, although there are explanations to be found in the literature.

(2) *Hyperplastic Ethmoiditis or Nonsuppurative Sinusitis.*— In the majority of cases we have found the same rarefying process of the middle turbinate and ethmoidal cells as in atrophic rhinitis. The mucous membrane covering this bone, however, becomes degenerated in the form of a pseudomyxoma, otherwise known as polypus. The usual accompaniment is excessive irritability of the nasal mucous membrane, which produces the marked sneezing usually followed by an excessive watery discharge. The pathologic change is not so marked in hay-fever, so far as rarefication is concerned, and we do not find any great changes in the mucosa unless the symptom of asthma is present. The large number of cases of so-called bronchial asthma that the otolaryngologist encounters have invariably the clinical picture of a nonsuppurative sinusitis. At first no polypi are visible, but later almost always. Histopathologically there is present the rarefying changes of a hyperplastic ethmoiditis. We believe, therefore, that these three conditions are due to the same cause, namely, a hyposecretion or disharmony of one or more of the glands mentioned above and that the underlying cause is a focal infection. We believe that the infection is most frequently from the intestinal tract; that these patients are sensitized particularly to protein or albumin and have attacks such as sneezing and asthma, which is a true anaphylaxis. That there is a deficiency in the glandular secretion may be explained by the fact that when we administer a large dose of adrenalin to a patient in the attack of asthma, he is frequently relieved. It has been asserted that this is due to the action of the drug, by raising the blood pressure, or, as an antispasmodic; but we believe it is a temporary satisfying of the hyposecretion. The treatment, of course, is to remove the original cause, namely, the focus of infection, and to administer glandular extract to temporarily satisfy the system. The local nasal pathology is taken care of. A middle turbinectomy, or

ethmoid exenteration, is as a rule sufficient. However, no permanent cure of any of the conditions mentioned can be obtained by any of these operative procedures alone, so far as we have observed.

(3) *Otosclerosis.*—About five years ago, while studying the histopathology of this disease on some specimens presented to me by Drs. Gray and Neumann, I noted the similarity of the spongifying process in otosclerosis to that of osteomalacia, arthritis deformans, and the bones in early months of pregnancy. I also found that experiments were being carried out on the treatment of these general processes by the administration of adrenalin, thymus, and pituitary extracts, and good results reported. We began the same therapeutic experiments on a few cases of otosclerosis, and made our report to the Southern Section of the American Otological, Laryngological and Rhinological Society at New Orleans in 1913. Since that time we have been employing these glandular extracts routinely in every case of suspicious or clearly diagnosed otosclerosis. Adrenalin has been the principal substance employed, although in a good many cases we combined the extracts of adrenal, thymus, and pituitary. Recently Dr. Smith of Delta, Colo., told me that he had been using the pituitrin exclusively with good results, and believed that it acts as an aid to the harmonic action of the other glands. Although we have the records of several cases in which there is no doubt that there has been improvement by this treatment, we have never maintained that we have cured a case of otosclerosis, or even improved the hearing. Again, we have the statements from otologists who have treated some cases by the use of adrenalin, of improvement in the hearing as well as in the tinnitus aurium. What we do assert is this: that cases of otosclerosis treated by injections of adrenalin 1:1000 solution from 1 to 15 minims in gradually increasing doses have shown a tendency not to progress. The injections should be controlled by measurement of the blood pressure and should be given every second or third day for a period of six weeks to three months; then interrupted for the same period of time; then resumed for the same period, and then stopped. We have, furthermore, removed foci of chronic infection, particularly from the tonsils and teeth, in such cases and obtained permanent relief. Most of these cases were not treated by us by any other methods, such as inflation of medicines. In some of these cases we administered also the pituitary and thymus gland extracts. As will be shown later in Dr. Pol-

lock's paper, long continued use of adrenalin extract is said to produce an arteriosclerosis, but in all these years we have seen no evidence of this affection clinically from such employment, and we have had no other untoward results.

About two years ago, before the Middle Section of the American Laryngological, Rhinological, and Otological Society, I reported on the value of roentgenography in otosclerosis, and stated that I was able to demonstrate the spongification process. I called attention to the use of this method to control the progress of the disease by repeating the roentgenography, thus determining whether there had been any further involvement.

(4) *Septum Deflections with Excrescence.*—We make no positive assertion that the etitologic factor in septum deformity is disturbance of the glands of internal secretion, but we do positively state that in the examination microscopically of deviated septa and excrescences we find the same rarefying process as in the turbinated bones, the internal ear, osteomalacia, etc. Since there is no definite etiologic factor for deviation and excrescences of the septum, may we not be permitted to theorize on the basis of true pathologic conditions in their analogy that disturbed internal secretions may have something to do with it? A practical point is, may it not be prevented from developing?

(5) *Exophthalmic Goiter or Thyrotoxic Conditions.*—This affection is treated by the neurologist, internist, general surgeon, ophthalmologist, and otolaryngologist because the symptomatology as well as the treatment is so varied and manifold. So far as the otolaryngologist is concerned, it is the recognition of the earliest symptoms that concerns him, as well as carrying out the real beneficial treatment. Many of these patients come to us with headaches and ear noises, and in many instances they have been improperly treated by operation, or treatment within the nasal cavity, or by inflations of the middle ear, none of which have proved of any permanent value. If one does recognize the other cardinal symptoms of hyperthyroidism as rapid pulse, fatigue with prostration on slight exertion, tremor, perspiration, diarrhea, and the fine soft skin, especially of the hands, whether there be exophthalmos or thyroid enlargement or not, one will be able to make the correct diagnosis. One or two doses of thyroid extract will markedly increase all the above symptoms, and the administration of the serum or extracts of thyroidectomized animals will relieve them, at least temporarily. The surgical treatment has held sway, and still

does, because the results are so striking from the ligation of the thyroid vessels with subsequent resection of part of the gland in the bad cases, and the immediate removal of part of the gland in the not far advanced cases. Our results from these procedures have shown us that they are not permanent and our observation of many cases operated on elsewhere show the same. We have, however, some of these recurrent cases, as well as many others not operated on, that were subjected to a tonsillectomy, that do show no tendency to recurrence. There is one case in our experience worth while mentioning very briefly, as it shows the value of tonsillectomy in this affection. I was called by a competent general surgeon and an equally competent internest to see a case of far advanced exophthalmos, to examine the tonsils with the idea that these might be infected, and, if so, to discover if they could be washed or treated, so as to improve the general condition of the patient, in order that a ligation of the thyroid vessel might be done. The resident physician gave me a wrong message, stating that I should come to the hospital and remove the tonsils from that patient. This I did, without any hesitancy, since I knew these gentlemen to be judges as to the indications for the removal of tonsils, and consequently did not even examine the throat before operation. There was nothing unusual about the operation, except that the patient was very nervous. When later in the day the surgeon and internist found out what had been done they became very much alarmed, and, of course, expected an exitus, but today, four years since the operation, the patient is permanently cured of the hyperthyroidism.

(6) *Myxedema.*—I have had two cases of myxedema in which there were marked laryngeal symptoms, in that the voice became very much changed. In one of them, that of a lady who previously had a pleasant high voice, the voice changed to a deep masculine type. There was nothing to be found except a suggestion of the cords being hypertrophied, but not inflamed. The other case was that of a man who was suffering from adenocarcinoma of the larynx and was subjected to thirty deep penetration radiotherapeutic treatments over the region of the thyroid, following the removal of the growth under suspension laryngoscopy. The symptoms of myxedema disappeared following the administration of thyroid extract, twenty grains daily, for six weeks. Another point of interest is the non-recurrence of the carcinoma (three years). The use of thyroid cured this condition permanently.

(7) *Status Thymicus or Status Lymphaticus.*—We have already stated that we believe the lack of thymus secretion has something to do with the rarefying changes in the bones and that we employ it in the treatment of such conditions. As to true diseases of this gland, we are most concerned in the lymphatic dyscrasias and the occasional pressure symptoms on the trachea. In all cases of infants in which we find marked glandular enlargement, particularly with enlargement of the tonsils and adenoids, we are suspicious that there is trouble with the thymus. Consequently we have, in the past eight years, not operated on any of these cases radically. We usually refrain from operating on them at all, and if a tonsillectomy and a partial adenectomy is performed, it is done without any anesthetic. We have, furthermore, adopted a rule not to operate on infants or children under 3 years of age unless extreme indications are present. We have been much criticised by the profession, especially some of the pediatricians, for this view, but we have the records of enough cases to show that it is not good practice. There are four fatalities among them. I operated on none of these patients myself, but I saw them in consultation. Three died within five weeks from symptoms resembling marasmus, and one very much like a cretin. We have seen a fair number of cases of infants that had tonsils and adenoids removed radically, following which there was a marked fattening of these babies, to the delight of the physicians and parents, but subsequently it was found that these children were somewhat backward in mentality and contracted colds and other diseases of infancy much more easily. On inquiry, we have seldom had similar experience expressed by other physicians.

Epilepsy.—This subject is of interest to the laryngologist because cases have a great many times been reported in the literature which presented certain growths within the nose and throat, such as sharp spurs on septum, adenoids, and tonsils, which when removed, cured the epilepsy. We have had a number of similar cases; but not in a single instance did we have any result from such treatment. I wish to call attention to the work of Dr. Crile, who reports marked improvement in grand mal following the removal of an adrenal gland. What is the cause of this hyperadrenalism? Is it a chronic focal infection? If so, where is it located?

The discussion of the hypophysis from our personal experience will be continued by Dr. Pollock.

In conclusion, I wish to apologize for the manner of presenting this important subject, which its vastness made necessary, but as to its timeliness I have no apologies to make.

NATURE AND CONTROL OF HEMORRHAGE IN NASOPHARYNGEAL OPERATIONS

JOSEPH WEINSTEIN, M.D.
NEW YORK CITY

The operative technic in nasopharyngeal work has recently seen such steady, though gradual, improvement that the procedures involved have at the present time reached a high degree of efficiency. Indeed, numerous methods offer themselves to the specialist in this field of surgery, and it is the ability to choose and execute the one procedure specially adapted to the exigencies of the case in hand that distinguishes the man with extensive and varied experience in nasopharyngeal work—the specialist—from the occasional operator on the nose and throat.

Such great progress in operative technic has had, however, one undesirable feature. It has somewhat blunted the recognition of the fact that nasopharyngeal operations are in many ways similar to the major operations of surgery, involving, as they do, comparable risks from shock, anesthesia, infection, etc.

Moreover, the one striking thing about a major operation is its comparative bloodlessness, and the great care taken to effect such bloodlessness to the highest degree possible. In nasopharyngeal work, on the other hand, much remains to be done to perfect this phase of the operation before any results that are comparable to those attained in general surgery can be secured. Yet careful comparison of a laparotomy with a tonsillectomy, let us say, shows that the dangers of uncontrolled hemorrhage, or oozing, are rather more marked in the latter than in the former. To be sure, bleeding from a large vessel is equally dangerous, whether the blood enters the peritoneal cavity or gushes from the mouth; and its prevention, that is, ligation of the large vessels, is equally imperative, and the procedure is about the same in either case.

However, smaller hemorrhages are of greater significance in our work, because the blood is apt to enter the bronchi and the lungs with much more serious consequences than if the free peritoneum were involved. Moreover, such blood is more apt to be, and indeed almost always is, mixed with infected tissues or secretions; for it is evident that no nasopharyngeal

field can be made as sterile as a clean peritoneal cavity, or other field for surgical interference. To the dangers of mechanical damage due to interference with respiration and with the circulation in the lungs, is added the probability of infection with all its dire consequences, from a slight bronchitis to a bronchopneumonia or a lung abscess. In this connection, the great difficulty of immobilizing the site of bleeding in nasopharyngeal work, the site of operation being usually involved in the movements of respiration, swallowing, vomiting, etc., must be remembered and compared with the greater ease with which other fields may be immobilized, from the complete rest of an extremity to the fairly complete paralysis of the intestines attainable by the use of drugs.

Yet there is no denying the fact that nasopharyngeal operations, in which these considerations are hardly given a thought, are done by the hundreds, and it is probable that it is merely the lack of such records as are usually available in the major surgical field that hides the fact of the extensive amount of damage that results.

There seems to be no reason why progress in this important detail of operation should not go on at a more rapid rate than heretofore. Careful study of the literature and extensive experience in special nasopharyngeal work have convinced the writer that there has been rather a laxness in the application of the various methods involved rather than absence of remedies dealing with the evil.

The procedures involved in dealing with nasopharyngeal hemorrhage are preventive, surgical, and medical. The preventive methods, in the first place, depend upon such study of of the general condition of the patient as may reveal any constitutional conditions favoring a hemorrhage, from the much feared hemophilia to diseased kidneys, with consequent high blood pressure. Such study we may safely leave to the internist, or to the family practioiner, who is usually the intermediary between the patient and the specialist surgeon. I would like to warn the latter, however, from taking any unnecessary risks in the matter with patients who reach him directly, or in which the general examination has not been thoroughly performed. The time spent in having such examination done, while apparently thrown away in case after case, may save him from the great misfortune of witnessing a serious or fatal outcome after

operation on a patient who should either not have come to operation, or who should have been properly prepared for the procedure.

Further prevention is inherent in such mastery of technic as shall result in the choice of the method best adapted to the individual case. In this connection, the recent anatomic studies of Balatnikow are interesting.[1] He says the topographic relations between the large vessels and the lateral pharyngeal wall are dependent, in a great measure, on the development of the bony parts of the skull in that region; and that, on account of the poor development of the visceral portion of the skull, the blood vessels lie closer to the tonsils in adults than in children.

The position of the head has an influence on the topography of the vessels and their relation to the tonsils. The blood vessels are closer to the lateral pharyngeal wall on that side from which the head is turned away, and are further away on the opposite side.

The distance between the tonsils and the internal carotid is about 1.5 cm., and between the tonsil and the external carotid artery is about 2 cm. This distance, however, is variable. Opening the mouth and depressing the tongue was found to have no influence on the position of the blood vessels in the dead body, but in the living it is possible by such maneuvers to bring about a change in the position of the vessels, particularly if there is an infiltration of the peritonsillar tissue. The pharyngeal wall is in intimate relation with the internal and external carotid arteries, the facial, the lingual, and the ascending palatine artery, and sometimes with the ascending pharyngeal artery; also with the submucous peripharyngeal plexus of veins. The bouquet of Riolan, a cluster of ligaments and muscles attached to the styloid process, cannot always be relied on as a protection to the blood vessels, as their relation is dependent on the height of the cut.

Speaking about hemorrhage, Balatnikow says: Hemorrhage following operation on a pharyngeal abscess may come either because the arteries are wounded or because the veins are distended or varicose. As the arterial branches reach only the tonsil capsule, breaking up into capillaries on entering the tissue of the tonsil, tonsillectomy should not be followed by more than capillary or venous bleeding. If the tonsil, however, is completely removed within its capsule, severe arterial bleed-

1. Arch. f. Laryngol. u. Rhinol., 1915, **29**, part 2, 225.

ing is possible. Because of the possibility of an abnormal position of the blood vessels, they should be carefully located by palpation before operating.

The following operating rules are laid down by Balatnikow:

In tonsillectomy, only that portion of the tonsil which projects into the throat should be removed. The tonsils should never be pulled out of their niche, and the instrument should never be pressed hard against the lateral wall of the pharynx.

In pharyngeal abscess, all precautions should be taken to check possible bleeding—instruments to sew the pillars, or even to ligate the common carotid artery, being held ready.

The patient's head should be firmly fixed, the medium position being suggested as the one most desirable both from the standpoint of danger and facility in operating.

The knife should only be used in making the incision through the mucous membrane. All deep exploration must be done with a dull instrument. The wound should not be carried more than one and one-half cm. deep if a sharp or pointed instrument is used.

The surface of the cut should be parallel with the long axis of the mouth, and the knife must never be directed backward or outward. The site of the incision is determined according to Thompson, i. e., the wound should be made in the apex of the external inferior quadrant, which is determined by drawing a line horizontally through the base of the uvula to meet a line drawn vertically through the anterior edge of the anterior faucial pillar.

The following hemostatic measures should be employed in the order given, in cases of postoperative hemorrhage following operation on the tonsils and lateral wall of the pharynx:

(1) Application of Mikulicz's compression forceps;
(2) Sewing together of the palatal arches;
(3) Compression of the common carotid artery;
(4) Ligation of the common carotid artery or its branches.

Also J. Leslie Davis'[2] investigations concerning the blood supply of the tonsils are of interest. He says: "There are certain fixed sources of all hemorrhages from tonsillectomy operations, and the majority of the textbooks are in error as to the blood supply of the tonsils." The entire supply comes from one artery, which is formed "by an anastomosis of the ascending palatine branch of the facial and the descending branch of the internal maxillary. The artery thus formed

2. Laryngoscope, March, 1914, **24**, 161.

enters the fossa at its superior extremity, passes downward through the capsule for a distance of about one-half an inch before penetrating the capsule to reach the tonsil parenchyma." The tonsillar branch of the facial artery, the branch from the dorsalis linguae, and the branch from the ascending pharyngeal supply only the surrounding structures in 95 per cent. of cases. Davis, therefore, after enucleation of the tonsil, places a suture ligature (using No. 1 catgut) around this artery in the upper part of the tonsillar fossa, and ties it off. This is his routine practice in all adult cases; but with children, only when the bleeding is profuse and continuous.

While some of the conclusions drawn by the above investigators may be open to criticism, it is the purpose of this paper to call your attention to the amount and character of the work that has been done, rather than to its merits or demerits.

Based on the commonly accepted causes of hemorrhage, viz., high blood pressure, persistent vasodilatation, and delayed blood clotting, therapeutic measures employed in the control of hemorrhage are of three kinds:

(1) Those equalizing the circulation and lessening the intravascular pressure at the bleeding point;

(2) Those contracting the vessel itself;

(3) Those decreasing the coagulation time of the blood.

Recent investigations of the phenomena of clotting of the blood have shown that normal blood in itself contains substances which, under certain conditions, unite to bring about coagulation.

According to Morawitz, a substance which he calls "thrombokinase" is present in all body tissues and especially in blood platelets. Prothrombin, antithrombin, calcium salts, and fibrinogen are constantly present in normal blood. When the tissues or formed elements of the blood are injured, thrombokinase combines with the calcium salts and prothrombin to produce a new substance called thrombin, which, with the fibrinogen, forms fibrin or clot.

According to the investigations of Bordet and LeLange, thrombin is formed by the reaction between cytozym and serozym in the presence of a calcium salt. The cytozym is found in platelets, leukocytes, tissue juices (probably the thrombokinase of Morawitz and Howell) and the serozym in the blood serum. These investigators attribute the formation of fibrin to the action of thrombin on fibrinogen.

Howell assumes that, while the elements of coagulation are all present in the blood, there is normally sufficient antithrombin to prevent clot formation. The balance between the two is very delicate. Cellular injury releases a substance which he calls thromboplastin, which neutralizes or binds the antithrombin and permits the other elements of the blood to combine in the formation of the clot. Howell's view seem to explain the known phenomena of coagulation more completely than the earlier theories.

While we cannot enter into a detailed review of these studies, the theories of the formation of antithrombin must be mentioned as being of great interest and importance to the intelligent understanding of the factors underlying operative hemorrhage.

Most investigators agree that antithrombin is formed in the liver. Experiments on the origin of antithrombin have been carried on, with few exceptions, with Witte peptone, which, when injected intravenously into dogs, produces an anticlotting substance in the blood. That the liver is at least a factor in the production of antithrombin has been shown by the discovery that in dogs in which the liver has been eliminated from the general circulation, injections of peptone do not have an anticlotting effect on the blood.

Denny and Minot[3] conducted a series of experiments to determine the antithrombin content of the blood in different parts of the body and attempted to produce antithrombin. Their investigations included a study of the effect of liver destroying substances on the antithrombin content of the blood and have led to the following conclusions:

Antithrombin is formed in the liver. Venous blood taken from the liver, spleen, kidneys, and intestines shows no appreciable difference from the blood of the jugular vein in antithrombin content.

When stasis is produced in any of these organs, only the liver blood shows a definite increase in antithrobin.

Perfusion of the liver with defibrinated blood causes an increase in antithrombin in the perfusate. Perfusion of the head, spleen, and hind leg shows no marked increase, and generally a decrease, in antithrombin content.

Attempts to stimulate the liver to production of antithrombin by means of bile, bile salts, secretin, and electric

3. Am. Jour. Physiol., August, 1915, p. 233.

stimulation were negative. Injection of small amounts of thrombin into the portal circulation caused a slight rise in antithrombin.

Dogs with phosphorus poisoning and liver destruction show a marked decrease in antithrombin content of the blood, as well as a diminution in fibrinogen.

An interesting paper by Katherine and Cecil Drinker[4] on the effect of rapid progressive hemorrhage on the coagulation time of blood was published in Feburary, 1914. According to these investigators, such hemorrhage causes a decrease in coagulation time, a gradual steady fall being noticeable as hemorrhage proceeds. This change was accompanied by a decrease in antithrombin, its amount remaining constant when the coagulation time was unchanged. Prothrombin was shown to increase slightly at first and then to decrease slightly. Fibrinogen was found to decrease as hemorrhage progressed. Platelet counts did not vary with progressive hemorrhage.

Gray and Lunt[5] conclude that moderately severe hemorrhage (13 per cent. of the circulating blood) decreases clotting time. The important coagulating factors may be localized in the abdomen because: (1) Exclusion of the abdominal circulation increases clotting time, and (2) Hemorrhage after exclusion no longer results in decrease of clotting time.

The adrenal glands probably favor, by their secretion, rapid clotting after hemorrhage; but after adrenalectomy, hemorrhage still decreases the clotting time.

Exclusion of the intestines usually increases clotting time; but, after this exclusion, hemorrhage can still decrease the clotting time.

The important rôle which the liver has been proved to play in coagulation indicates that it provides, perhaps in cooperation with the intestines and the adrenal glands, the necessary elements to hasten clotting after extensive bleeding.

Vosburgh and Richards[6] in 1903 noticed that an increase in sugar content of the blood caused the blood to coagulate more rapidly. They obtained this result by painting adrenalin on the pancreas, and concluded that this was due to the action of adrenalin on the pancreas. A few years later, Wiggers discredited this. Von den Velden[7] in 1911 asserted that adrenalin

4. Am. Jour. of Physiol., February, 1915, p. 305.
5. Am. Jour. of Physiol., June, 1914, p. 332.
6. Am. Jour. of Physiol., 1903, **9**, p. 39.
7. München. med. Wchnschr., 1911, **58**, p. 187.

decreased the coagulation time in man about one-half, but attributed it to vasoconstriction which caused a discharge of tissue juices into the blood.

Cannon and Gray[8] discredit the theories of both Vosburgh and Richards and of von den Velden, and conclude that:

Adrenalin injected in small doses intravenously (0.001 mg. per kilo) and in large doses subcutaneously, will shorten coagulation time to one-half or one-third the former duration. The prompt shortening of the process after small doses is changed after larger doses (about 0.03 mg. per kilo) to a lengthening and later a shortening, or to a lengthening alone.

The effect of adrenalin on the clotting time is not associated with any corresponding effect on arterial pressure.

If the blood is confined anterior to the diaphragm, or if the intestines and liver are removed, adrenalin in small doses does not cause rapid clotting.

The addition of small amounts of adrenalin to drawn blood does not hasten clotting. Increase of dextrose in the blood to 0.3 or 0.4 per cent. does not cause the rapid clotting seen after adrenalin injection.

The explanation is suggested that adrenalin accelerates the clotting process by stimulating the liver, and perhaps the intestines, to greater activity in discharging some factor or factors in coagulation.

In the same year, Cannon and Mendenhall, using as a basis for their experiments the discovery that injected adrenalin shortened the coagulation time of blood, attempted to increase the secretion of the adrenals by stimulating the splanchnic nerves to produce similar effects, and secured the following results:

Stimulation of the splanchnic nerves results immediately, or after a brief delay, in shortening the coagulation time of blood.

Such stimulation usually produced less marked effect when it was repeated. If the adrenal gland is removed on one side, splanchnic stimulation on that side does not shorten clotting time; whereas, splanchnic stimulation on the other side is still effective. The faster clotting is therefore due to increased adrenal discharge.

Since stimulation of the nerves supplying the liver and intestines does not hasten clotting, and since increase of adrenalin has no effect in the absence of the liver and intestines, the short-

8. Am. Jour. of Physiol., May, 1914, p. 232.

ened clotting after splanchnic stimulation is accounted for by the action of adrenal discharge on the liver and perhaps the intestines.

In a study of the effects of pain and emotional excitement on coagulation of the blood, these same investigators conclude that:

Stimulation of afferent nerves (sciatic, crural), or major operations under light anesthesia, markedly shorten the coagulation time of blood. Emotional excitement is the occasion for very rapid clotting, which becomes slow when the splanchnic nerves are cut. Pain and strong emotions have been proved to evoke secretion of the adrenal glands; and adrenalin hastens clotting. Rapid coagulation may be reasonably considered, therefore, as another instance of adaptive reaction serviceable to the organism in the injury which may accompany pain, or which may follow the struggle that fear or rage may occasion.

Before concluding this part of my paper, I wish to call your attention to some recent investigations of the effects of chloroform and ether on the coagulation of the blood. In July, 1915, Mendenhall[9] published a report of his investigations on this subject, from which he drew the following conclusions:

Coagulation time is little altered by chloral hydrate.

Coagulation time is affected by chloroform as by chloral hydrate. The effects of chloral hydrate and chloroform are probably the result of disturbance and consequent interaction between two or more organs which are important in the coagulation process; probably liver, intestines, and adrenal glands.

The evidence is not sufficient to prove that a retarding agent is produced.

Coagulation processes are hastened by ether anesthesia. The effect of ether is exerted wholly through its action upon the adrenals.

In December, 1915, Minot[10] summarized his investigations as follows:

Antithrombin is rendered inactive by chloroform and ether, thus allowing free thrombin, if present in an oxalated plasma, clot fibrinogen.

Prothrombin is not converted into thrombin by chloroform. Chloroform can precipitate both fibrinogen and prothrombin from an oxalated plasma. Chloroform does not weaken the action of a solution of pure thrombin. Ether does slightly.

---

9. Am. Jour. of Physiol., July, 1915, p. 33.
10. Am. Jour. of Physiol., December, 1915, p. 131.

Antithrombin could not be recovered from chloroform or ether extracts of serum or plasma, unheated or heated to 60 degrees C., and is not exactly identical with antitrypsin, or with Doyan's antithrombin.

In one chloroform poisoned rabbit, the antithrombin of the blood was decreased below normal.

Having in mind the conclusions of these and other investigators in the field of physiology, other scientists in the fields of biochemistry and pharmacology, have been at work with the idea to strengthen and hasten, by therapeutic means, the natural processes of coagulation. I shall mention a few of the better known products which have been the result of their efforts:

Immediately after, Oliver and Schaefer, and Czybulski and Szymonivicz discovered in 1895 that there was a very marked rise in blood pressure after intravenous injections of extracts of the suprarenal glands, Moore demonstrated "that the active substance was only present in the medullary portion of the gland and that it was identical with a chromogenic substance, described by Vulpian as early as 1856, possessing striking color reactions—green coloration with iron chlorid on the addition of alkalies, or with iodin or chlorin water a pink-carmine color. The reaction suggested benzecatechin and von Furth succeeded in preparing from this chromogen a substance which in its behavior agreed with benzecatechin. Takamine first prepared the crystallized active substance in 1901 and named it adrenaline." (Meyer and Gottlieb.) To discuss further so well known a substance as adrenalin would be superfluous.

In 1867 Pelletier separated from ipecac its emetic principle, which he named emetin. In 1894, Paul and Cownley isolated its two crystalline alkaloids, emetin and cephaëlin. The uses to which this drug has been put are also well known. My personal experience with emetin in nasopharyngeal hemorrhage has been extremely gratifying. While I am unable to say in what manner it acts, I have come to look on the injection before operation of one-half grain of emetin as a routine procedure in my operative work. I made a close study of my results when I first began to use emetin, using the Duke coagulometer in making my tests. My results after several months' experience warrant me in asserting that by a hypodermatic injection, fifteen minutes before making my tests, of one-half grain of emetin, I am able to reduce the bleeding time one-third at least.

Pituitrin is described as an extract made from the infundibular portion of the hypophysis. In 1913, Citelli published a report of his gratifying experiences with pituitrin in the control of nasopharyngeal hemorrhage; and since then there have been other reports of a similar character. I tried out pituitrin in the same manner as I did emetin and obtained practically the same result, i. e., the bleeding time was reduced about one-third. I did not, however, get as uniform results as I did with emetin.

Coagulum ciba was discovered by Kocher of Berne and his assistant, Dr. Anton Fonio, and is described by them as an extract of blood platelets containing cytozym, which, when added to animal blood, combines with serozym existing in the blood and forms thrombin or fibrin ferment. Being wholly within the bounds of a physiologic process, coagulation with this preparation is regarded as harmless and nonirritating.

Fonio has published his personal experience in the treatment of 700 cases and says that in no case was any septic effect observed. Kausch, after treating 300 cases with coagulum ciba, confirmed Dr. Fonio's experience, as did also Dr. Obermueller, after an extensive use of it in rhinologic practice. My own experience with this product has not been so extensive as with the two former preparations; but in every case in which I have used it, it has shortened the bleeding time; in a number of instances having had an almost instantaneous effect.

Thromboplastin (tissue juice) is prepared from the liver and brain of cattle. Dr. Hess[11] published in April, 1915, his preliminary report of his experience with this tissue juice, and since that time other reports have been made of its successful use in the control of hemorrhage. Dr. John T. Cronin of the New York Department of Health reports on its use as follows:

The use of thromboplastin has been extended to all Department of Health nose and throat clinics. In a summary of 2,036 tonsil and adenoid operations, the results were all excellent; in a large majority of the cases, there was no bleeding at all and in the remaining cases only slight bleeding. There were no bad after effects. In no case has the operator or nurse been obliged to return to the clinic to care for a bleeder, since the use of thromboplastin has been made a routine procedure.

In the comparatively few cases in which I have used thromboplastin, I have had uniformly good results.

---

11. Jour. Am. Med. Assn., April 24, 1915, p. 1395.

Coagulose, a product obtained by precipitating normal blood, and said to contain the fibrin ferment necessary for clotting blood, is reported to have given good results in nasopharyngeal hemorrhage. Frey in 1898 treated hemorrhage with normal horse serum. Weil reports a case in which he, by means of this treatment, shortened the coagulation time from four and one-half hours to forty minutes.

Still other products have been offered from time to time for the control of hemorrhage, but have not become so well known and their effect has, consequently, not been so closely studied.

I have attempted to show that recent investigations by anatomists, physiologists, and pharmacologists have a direct bearing on the daily problem of our work; and it is no less than imperative that the results obtained by these workers should find prompt application and control in actual operative technic. Such course of action must require a somewhat different attitude toward nasopharyngeal surgery from that usually encountered. As I have shown, even the single element of hemorrhage presents as perplexing problems as any that may arise in the usual procedures of major surgery. It demands the same attitude from those of us who are workers in this special field that the pioneer surgeons and the present leaders of surgical science have taken in their work.

Such an attitude, and with it success in the development of this important branch of medical endeavor, must rest on the following factors:

Careful study of the local anatomic situation and the discovery of facts leading to the improvement of general technic and especially improvements in the prevention and treatment of hemorrhage.

General examination of the patient for the purpose of discovering any constitutional or morbid factors leading to the decrease in the clotting index of the blood.

The choice of proper anesthetic and its proper administration.

The study of new substances developed in the laboratory and applicable for the diminution of the tendency to bleeding and in checking such hemorrhage, once it has set in.

And, finally, such cooperation between the different workers in these varied fields as shall most certainly and quickly lead us to our goal, that is, the proper and efficient correction of morbid conditions of the throat and nose, attained with the least discomfort and danger to the patient.

THE INTERPRETATION OF STEREO-ROENTGENOGRAMS OF THE MASTOID

J. M. Ingersoll, A.M., M.D.
Professor of Otolaryngology in the Medical Department of Western Reserve University, Cleveland, Ohio

The practical value of stereo-roentgenograms of the head depends upon the correct interpretation of the stereoscopic picture. The only way to acquire skill in interpretating the picture is by practice and study. Fortunately it does not require the expenditure of much time or study to gain a reasonable amount of skill in interpreting the stereoscopic plates.

In order to identify positively in the stereoscopic pictures some of the landmarks in the bones of the skull, we took a macerated and cleaned skull, sawed off the top, uncovered the semicircular canals in one mastoid and inserted copper wires in the canals. Then we took stereo-roentgenograms of both mastoids of this skull, using the same technic that we use in the living subject. These plates gave us beautiful stereoscopic pictures of the mastoids, and the copper wires located absolutely the position of the semicircular canals. By studying these pictures and the skull alternately, the following structures can be definitely located: the external and internal auditory canals, all of the mastoid cells, the mastoid antrum, the semicircular canals, the groove of the lateral sinus and the jugular bulb, forming the floor of the tympanic cavity, the foramen lacerum posterior or jugular foramen, the foramen ovale, just anterior to the petrous portion of the temporal bone, the foramen lacerum medium showing dimly through the extreme anterior part of the petrous bone, the groove of the superior petrosal sinus on the posterior-superior surface of the petrous bone, the foramen magnum, the grooves of the middle meningeal arteries, and the floor of the middle fossa of the brain.

The plates should be placed in the stereoscope first, so as to show the mastoid from the outside; that is, with the film side toward the observer. In this way the temporal bone is seen with its external structures in the foreground and the the deeper structures in the background. Then by reversing the plates in the stereoscope, turning the smooth side of the plate toward the observer, keeping the right hand plate on the right side and the left hand plate on the left side, the mastoid is seen from the inside of the skull.

When stereoscopic plates of the mastoid in the living subject are examined they should be studied in the same way. First, looking at the plates from the outside, we can see the mastoid just as we shall see it later in the operation, with the additional advantage of actually being able to look into and through the bone. The size of the mastoid can be accurately determined, and we can see whether or not there are any cells extending forward into the root of the zygoma. The location of the lateral sinus can be clearly seen, and its depth below the cortex can be determined with considerable accuracy. The size and position of the mastoid antrum can usually be definitely seen. The amount and character of the infection in the mastoid can be determined.

Normal mastoid cells transmit the Roentgen-rays better than inflamed cells and slightly inflamed cells transmit the rays better than cells filled with granulation tissue and pus; hence, in normal mastoids, the cells and their walls and the underlying structures can all be distinctly seen. When there is only a mild inflammation in the cells, as in the early stage of acute otitis media, the cells all look dimmer and less distinct than they do in the normal mastoid. If the mastoid antrum and cells are filled with granulations and pus, the area thus involved shows much less distinctly on the plates. If the walls of the cells have been broken down by the suppuration and are necrotic, then the pictures show an exceedingly dull area which looks very much like an irregular foramen.

In other words, the stereoscopic plates show that there is a mild inflammation in the mastoid, or that there is pus and granulation tissue in it, or that there are areas of necrosis and that the sinus or dura are probably exposed.

Abscesses in the brain apparently transmit the Roentgen-rays about the same as the brain tissue does, and so do not, in most cases, cast a shadow that is definite enough to differentiate them from the brain tissue. We have had, however, several cases in which areas of slight dullness in the brain have suggested brain abscess and the operative findings have confirmed the diagnosis. In some of these cases we have taken a second stereo-roentgenogram before the gauze packing was removed from the abscess cavity and thus have been able to definitely locate the position of the brain abscess and verify the diagnosis of probable brain abscess, made on account of the clinical symptoms and the suspicious areas of dulness in plates taken before the operation.

By careful study and comparison of such plates, made before and after the operation, much valuable information can be gained

and we believe that, as our skill and experience in interpreting the plates increase, we shall be able to diagnosticate brain abscesses with increased certainty.

Stereo-roentgenograms of the mastoid give us not only much valuable information about our operative field in cases which must be operated on; but they also help to determine whether or not an operation is necessary.

In cases of acute otitis media, with pain over the mastoid antrum and tip, a stereo-roentgenogram helps very much in determining whether or not it is advisable to operate immediately. If the stereoscopic plates show only a little dimming, compared with the normal, with no definite dull areas and the cell walls can be distinctly seen, then we may safely keep the patient under observation, for the inflammation in the mastoid will probably subside. If the plates show a pneumatic mastoid, or distinctly dull areas in the mastoid, especially in the tip, and some of the cell walls have disappeared, such a patient will probably never recover without an operation, and the sooner the operation is done the better the prognosis.

So, too, in chronic otitis media, the stereoscopic plates help to determine the necessity for operative treatment. If the plates show that only the middle ear and mastoid antrum are involved, there is a fair chance of curing such cases by attic irrigation and local treatment. If, however, the plates show a cholesteatoma in the antrum, casting a dense black shadow, or involvement of the cells around and below the antrum, or areas of necrosis, such a patient should be operated on immediately.

Usually in chronic suppurative otitis media the whole mastoid becomes sclerotic, most of the cells disappear, and the stereoscopic plates show the area of involvement in and around the antrum, while the rest of the mastoid looks almost as dense as the petrous portion of the temporal bone.

If the plates show such a sclerotic process and there are no cells remaining in the inferior part of the mastoid, it is not necessary to go down into the tip of the mastoid in a radical operation, but remove only as much of the mastoid as is needed for working space in the antrum and middle ear. Such cases naturally recover more quickly than those in which a more extensive operation is necessary.

SUMMARY

Stereoscopic roentgenograms of the mastoid give us much valuable, accurate information in regard to the mastoid which

cannot be obtained in any other way. They locate definitely the anatomic structures in the operative field. They show the extent and character of the pathologic process. They show areas of necrosis in the bone, if there are any. They sometimes show the location of a brain abscess. They help to determine whether or not it is necessary to operate in doubtful cases.

I wish to thank Dr. Hill and Dr. Thomas for their interest in this work and the many valuable suggestions which they have made.

DISCUSSION

Dr. J. C. Beck, Chicago: It is a great pleasure to discuss a paper of Dr. Ingersoll's at any time, and particularly on this subject, in which I have been intensely interested for a good many years. About ten years ago, when I published my atlas on this subject, I inserted about six pictures of stereoroentgenograms of the mastoids and sinuses, and said then that I did not think they were of any particular value, that it was an unnecessary expense, and that at that time there was a possibility of injury, to some extent, because of falling out of the hair. Since that time I have changed my belief radically, so that scarcely ever do I use a single picture. If there is a picture necessary at all, the proposition of using the Roentgen-ray for the diagnosis and indication for operation is the important part that Dr. Ingersoll brought out. And while all these details are shown in the roentgenogram of the skull, especially if you insert wires through these canals, and so forth (and it is a wonderful study), it is not of any practical value. But the studying of roentgenograms of the living subject and the study of pathologic entities, that is a problem. You cannot expect a roentgenologist to tell you what the indications are in a given picture, nor can you tell until you have studied the subject of densities of different tissues, not only of normal, but of pathologic tissues, and know whether the indications are of a foreign body or sequestrum, or whether they are syphilitic, tuberculous, necrotic, etc. That requires much study, and many, many pictures are required to give all the points. I look forward with great pleasure to the time when a course shall be given in which this is taught; for there is certainly no other means of diagnosis of so much value in sinuses, mastoid diseases, and diseases of the middle ear. It was fine to see the technic. Most roentgenologists do not know this. I saw pictures here the other day of a frontal sinus showing an entirely faulty technic. They presented a displacement of the head and did not correspond to the proper technic, which should show the normal position, so that we can study the pathologic entites. We should have team work between roentgenologists and otologists. They should study the findings together. The technic is a very important part of the proposition. Dr. Ingersoll called attention to the value of looking at the head from both sides, which is known as stereoscopy and pseudoscopy. It is like looking into a glass ball and seeing inside of it the structure of the petrous portion of the temporal bones and the details of the mastoids; that is, in a true stereoroentgenogram. It takes practice to properly study stereoscopic pictures. The diagnosis also of marked pathologic conditions in the ear is possible.. You can see not only the destruction, but you can differentiate how far the destruction has gone. As to the diagnosis of brain abscess, and I have looked at a number of pictures, I have seen one that gave an indication which proved to be a brain abscess, in which the bone of the

temporal region was affected, and a study of the change in bone gave us some idea of the abscess. Dr. Ingersoll brought up the point of packing gauze which would be radio-obstructive. Saturating the gauze with iron or bismuth would aid still further in bringing out the desired detail. I have injected some skulls with bismuth paste, which makes very beautiful stereoscopic pictures for that purpose. Dr. Ingersoll called attention to the value of the mastoid picture, and said so long as the mastoid is clear you need not consider an operation. I think, so far as that is concerned, we may have a clear mastoid process and still we shall have to operate. There may be an irritating condition on the labyrinth or facial nerve which will require such a procedure. As to the indications for operation in chronic suppuration, I do not place so much value on these as Dr. Ingersoll does. I think in most of these chronic cases we have sclerosis of the mastoid, and the details are not easily discernible. And as to not cleaning out the entire process because it is dense, I take issue there, for it may be that the bone is infiltrated with cholesteatoma. That is a procedure that has been tried out by Heath and many others, and it has been universally rejected in this country. A radical operation, whether the mastoid bone is dense or not, means cleaning it out to the bottom. Nothing was said about sinuses. I had hoped to hear something of sinuses, but certainly this has been a treat for me and I thank Dr. Ingersoll.

Dr. Ingersoll (closing discussion): The picture of a macerated skull is valuable, because it gives you landmarks and points of orientation. I am perfectly free to confess that I was not able to recognize the foramen lacerum medium in the pictures of living subjects, until after I had studied the picture of the skull alone. The foramina and the grooves of the superior petrosal sinuses, for instance, I find now in some pictures of the living. You can take the skull and the picture and, studying them alternately, pick out the landmarks. That is why the wires were used: to aid in orientation and to give a stereoroentgenogram anatomy, so to speak. It helps you to interpret the living picture, if you are once familiar with a picture of a macerated skull. What Dr. Beck says about team work between the roentgenologists and the otologists is very true. I am fortunate to be in the same building with Drs. Hill and Thomas, who are exceptionally good roentgenologists. They have furnished me with many valuable suggestions and have also shown an interest, which has made the work valuable to me. Seeing the temporal bone from both sides, reversing the plates, is like taking the temporal bone in your own hand before you operate on it, and looking at it first from the operative side and then looking at it from the inside, and with the added advantage, too, of being able to look at it not only from both sides, but also through it. What Dr. Beck says about operating in acute otitis media with some symptoms of mastoid involvement, or involvement of the internal ear, is, I admit, perfectly true. The point I meant to make in my paper is, that in some cases of acute otitis media you get pain not only over the antrum, but also over the tip, and you are in doubt whether the mastoid is involved or not. If you take a picture and find the mastoid is a little dim, compared with the normal, and that there are no decided dark areas in it showing foci of infection, I say it is safe to keep that case under observation. You may have to operate later, particularly if the roentgenogram shows you that the mastoid has comparatively small cells. I said, if it shows large pneumatic cells, we know in that type of mastoid that the infection is likely to spread much more rapidly. Such a case should be regarded with more suspicion than a dim one with comparatively small cells. In chronic otitis media I still adhere to what I said: That if the

mastoid bone is sclerotic and there are no cells left in the tip, in doing the radical mastoid all that is necessary is to uncover the antrum and the middle ear and the region around it. Do a radical without going down into the tip. Remove the canal wall, open up the middle ear, but do not go down into the tip if there are no cells there. I have operated on quite a number of patients, and I have yet to operate on one the second time because of some focus of infection being left. The advantage in such cases is, a smaller cavity is left and the patients recover more rapidly.

THE SURGERY OF THE TEMPOROMANDIBULAR ARTICULATION

L. W. Dean, M.D., and W. F. Boiler, M.D.
IOWA CITY, IOWA

The proximity of the temporomandibular joint to the middle and external ear makes it of particular interest to the otologist. The fact that it is so frequently involved in diseases of the tonsils and of the peritonsillar structures and in dento-alveolitis, makes it of great importance to the laryngologist.

It is not unusual for patients to come complaining of sore throat, accompanied by a disturbance of occlusion, and feeling that the whole trouble is in the throat, when a roentgenogram of the temporomandibular joint will show a collection of fluid between the condyle and the socket. This condition may be secondary to a throat lesion, or the result of a focus of infection in some other part of the body. In our experience the patient usually refers the trouble to the ear; but the roentgenogram, in the presence of an apparently normal ear, demonstrates the trouble to be in the temporomandibular joint.

It is not our intention to review the surgery of the temporomandibular joint. Murphy and Kreuscher[1] have recently published in the February number, 1916, of *The Dental Cosmos*, a comprehensive review of the matter, and we anticipate you are familiar with this report. Following such an exhaustive treatment of the subject, it would be a waste of your time to have the matter again reviewed. Consequently, we are going to confine the paper to a report of several cases that we have had during the past year, and which have presented some very unusual features in derangements of the temporomandibular joint.

Report of a case of bilateral complete true bony ankylosis of the temporomandibular articulation which was either congenital or acquired immediately after birth.

The case was one of both true and false ankylosis, in that there was an osseous ankylosis of the joint itself—the true ankylosis. In addition to this, there was cicatricial tissue between the neck and ramus of the bone and the surrounding

1. John B. Murphy and Phillip H. Kreuscher: Ankylosis of the Jaw, The Dental Cosmos, February, 1916, **58**, No. 2.

tissues, and between the coronoid process and the zygoma and neighboring structures, producing a false ankylosis. In our experience, almost every case of true ankylosis has associated with it a false ankylosis.

The condition in which the mandible cannot be moved a normal distance apart from the maxillae is known as ankylosis. Brophy, quoting Blair's statistics, says they are in accordance with his own observations.[2] In his series trauma was the most common single cause. Nearly 50 per cent. of all cases were due to this. A blow or a fall on the chin was the most frequent single trauma. He places scarlatina as the second factor causing ankylosis. Suppuration following this disease accounts for about 20 per cent. of the cases. The following diseases play a part in the order named: Otitis media, dento-alveolar abscess, typhoid fever, pneumonia, measles, variola, diphtheria, and rheumatoid arthritis.

So-called congenital temporomandibular ankylosis is very unusual; also that due to trauma at birth or during delivery. The prevalence of ankylosis of the mandible during childhood appears logical, when we consider that accidents and many of the infectious diseases to which ankylosis is due, are more prevalent during the early period of life.

Ankylosis of the joint may appear soon after the trauma or many months later.

The obstetric forceps may cause fracture of the base of the cranium, or of the mandible near the joint in the new-born, and ankylosis may follow. Such cases may be mistaken for congenital ankylosis of the mandible.

Case 1.—A. E., female, white, aged 6, was referred by Dr. J. D. Cantwell, Davenport, Iowa. The entrance complaint was lower jaw trouble. The child had had mandible retracted since birth and had never been able to open the mouth at any time more than one-eighth of an inch. Early in life, the child was not able to nurse properly, and the mother attempted to force the mouth open by using her finger as a wedge. In this she was not successful.

The mother was a bleeder and during the birth of the two preceding children, she had had a great deal of trouble, and at the last delivery had almost died. During the delivery of the child under consideration, owing to very poor heart action and very little strength, it was necessary after prolonged labor to apply the forceps.

2. Brophy's Oral Surgery, p. 454.

The attending physician discovered the ankylosis immediately after birth. He examined the child at the time of birth, and his impression at that time was that the trouble was congenital. During the first eight months of the child's life she cried almost continuously. She had mumps when 4 years of age, and measles at 5.

Examination.—The mandible was retracted and ankylosed. The amount of movement of mandible was enough to clear the teeth about one-eighth of an inch. The child throughout her life had never been able to insert even the tip of the tongue between the incisor teeth. The angles of the jaw were laterally very prominent and there are deep indentations inferiorly just anterior to the angles. The mandible was very poorly developed and there was an overhanging bite of full three-fourths of an inch.

The skull, bones of the face, and superior maxillae were well developed.

The teeth were reported on as follows by Dr. Fenton: Upper lateral temporary incisors are not present. Radiograms show permanent lateral incisors in alveolar process. Upper temporary right and left cuspids are badly decayed and pulps are dead. Lower left temporary lateral is not present.

Diagnosis.—Dead pulps in upper cuspids.

Treatment.—Extract.

Unfortunately prints from these plates do not show the changes well. These can be seen properly only by means of a stereoscopic examination.

Mandible.—Right. Body was very thin, so much so that anteriorly there was only a paper plate of bone between the unerupted teeth and the margin of the jaw. The coronoid process appeared with the apex above the upper margin of the zygoma. In front of the angle of the jaw, the body was bent obliquely downward. Just in front of the angle was the first permanent molar. Above it and high up in the ramus, really in the body of the coronoid process was the second molar bud. The condyle seemed to be crushed. The top was blunt and in socket. The eminentia articularis could hardly be seen and is apparently flattened.

Mandible.—Left. Appearance of body, ramus, and coronoid process was the same as right. The condyle seemed fractured, pushed down and back, being higher and further back than normal. Condyle fitted into a square socket.

Diagnosis.—There was a fracture of the neck of the condyle with crushing of the condyle on each side; also a fracture of the angle on each side with mandibular ankylosis, bilateral and true bony.

Operation.—Resection of the neck of the condyle was performed (following the procedure as described by Murphy in 1902), with resection of the coronoid process of the mandible left.

Ether Anesthesia.—The joint was exposed by making an incision one and one-eighth inches long beginning just in front of the ear and extending along the superior border of the zygoma. From the anterior end of this incision, a short incision about one-half of an inch long was made upward and forward, so as to avoid injuring the temporal and orbicular branches of the facial nerve. At the posterior end of the incision, a second incision perpendicular to the first, and about two inches long, was made. The edges of the wound were drawn downward, the lower lip being displaced down below the lower border of the zygoma. The tissues were displaced downward until the neck of the condyle was exposed. The skin and the tissues in the lower part of the wound were tightly retracted, using a blunt retractor without injuring in the least the facial nerve. The articulation was fully exposed and no evidence of a joint could be discovered, the head of the mandible seeming to fuse with the socket in the temporal bone. Using a wedge between the jaws to tell when there was any loosening of the joint, the neck of the condyle was attacked with chisel and hammer. A V-shaped groove was chiseled across the neck and pieces of bone removed with small rongeurs. This was continued until the wedge indicated that the bone was divided. The wedge used by Dr. Fenton was especially constructed of very dense wood, and was employed as a pry to tell when we had a movable mandible. When the mandible was loosened, using rongeurs, the condyle and the upper part of the neck were removed. No protector was used for the internal maxillary artery, none seeming to be necessary. The small vessels were ligated and the hemorrhage controlled with hot water and adrenalin.

The next step was the preparation of the fascial flap. The triangular flap of skin outlined by the incision was elevated. A U-shaped flap of fascia and muscle, 1.5 inches wide and 2.5 inches long, having its base at the upper margin of the zygoma, was elevated from the surface of the temporal muscle. The flap was freed from above downward. It was left attached to the zygoma, folded down into the wound, the lower end being tucked down underneath the upper margin of the ramus. The flap was held in position by means of two catgut sutures, one placed at the antero-inferior angle, and one at the postero-inferior angle of the flap.

The coronoid process which was underneath the zygoma was found bound tightly to the zygoma, and it was exsected before the flap was placed in position. The jaws were now widely separted by Dr. Fenton by means of a plastic wedge of gutta-percha placed between the teeth of the right side and fixed in position. This was done to prevent the upper end of the ramus of the jaw from pressing the flap and causing necrosis. The skin wound was closed, tincture of iodin was applied to the wound, no other dressing being used. This operation was performed on Aug. 6, 1916.

On Aug. 23, 1916, a similar operation was performed on the left side. So far as we could determine, there was no involvement of any of the branches of the facial nerve following either operation.

About three weeks after the operation on the right side there was some infection underneath the skin on that side. The skin was incised and a very slight amount of pus escaped.

The healing of the left side was uneventful. After the operation on the left side was completed the teeth were held apart about 1.5 inches by the two gutta-percha wedges which Dr. Fenton had placed, one on the right and one on the left side.

It was interesting to watch the little child lie in bed, protrude her tongue, draw it in and protrude it again. It was an amusing thing to the child, because she had never been able to even

Photograph of A. E. before and after operation.

insert the tongue between the teeth. The mother said she had never before seen the child's tongue.

On Aug. 30, 1916, that is, one week following the operation on the left side, Dr. Fenton removed the two wedges of gutta-percha from between the teeth. At this time, the patient could move the jaw nicely, so as to open the mouth 1.5 cm. The mouth could be shut tight. The jaws could be closed so that the occlusion was fairly good.

At this time, Dr. Fenton began daily passive exercises, using a wedge to pry the mouth open. These exercises were continued for two months, being given daily. At this time the patient went home. The patient could open and close the mouth nicely. The opening was about two-thirds that of the normal.

In connection with the operation for ankylosis of the temporomandibular articulation, we would like to make one point, namely, always make the fascial flap much longer than seems necessary in order that it can be tucked down in the wound without any tension on its attachment to the zygoma.

Case 2.—H. L., white, male, aged 50, came under my observation on Aug. 22, 1916. Entrance complaint was ununited fracture of the right side of the mandible, with inability to move the mandible.

On April 23, 1916, the patient was kicked on the right side of the lower jaw by a horse. There was a comminuted fracture anterior to the angle. This was wired at the time. The patient came to us with an ununited fracture.

Examination showed that the fractured ends of the bone on the right side were widely separated. The posterior fragment extended laterally beyond the anterior. There was nothing about the fracture to suggest why the left side of the mandible was immobile and why the jaw was kept in the open position. Inquiry failed to reveal any history of injury on the left side.

Roentgen-ray examination revealed on the right side, as shown in Exhibit 3, fluid in the temporomandibular socket. The margins of the fragments were widely separated.

Exhibit 4 shows the findings on the left side. An old fracture of the neck was discovered.

The diagnosis was: Ununited fracture of the body of the mandible on the right side anterior to the angle. Healed fracture of the neck on the left side with true bony ankylosis.

With this patient we felt that we had to deal with a very difficult problem indeed. In order to get a good result here we felt that it was necessary to bring the parts in apposition, hold them there by some appliance, and keep the mandible at rest until healing took place.

We had discovered also a true bony anklylosis on the left side. In order to get a good result here, we thought it would be necessary to exsect the condyle, make a fascial flap and then exercise by passive motion this new joint for a number of weeks. The matter was explained to the patient, and we advised first, the correction of the ankylosed joint. This was to be followed weeks later, if necessary, by a correction of the ununited fracture.

The operation previously described was performed on the left side. One week later, the exercise of the mandible with the wedge was begun.

One week following the operation, when the gutta percha wedge was taken out, the patient had good occlusion. The motion of the mandible was a little over 0.5 inch. The passive motion was continued for five minutes daily during three or four weeks, at

the end of which time the patient could open and close his mouth in a normal manner.

Following the exsection of the neck on the left side, the pieces at the point of the old fracture were brought in fairly good apposition. Much to our surprise, without any operative procedure or appliance being applied to the point of fracture, and with the daily passive exercises in order to secure motion of the joint, the ununited fracture healed, giving the patient a fairly good result without a second operation.

Report of a Case of Bilateral Forward Dislocation of the Mandible of Almost Five Months' Standing Reduced by Manipulation.—The interesting thing in connection with this case is the length of time that the dislocation existed before it was reduced. The dislocation occurred in September, 1915. At that time the family physician, Dr. Doering of Fort Madison, Iowa, wrote that he had a patient with a double dislocation of the mandible, and that he would like to have her come at once for treatment. For some reason or other the patient postponed her visit to us until a period of almost five months had passed. We had come to the conclusion that the patient did not have a dislocation of the mandible, because we would not conceive of a patient with a condition like this not having something done.

What was our surprise, when, at the end of this time, the patient presented herself at our office with the open mouth and all of the characteristic symptoms of a bilateral dislocation of the mandible.

Brophy[3] reports that May successfully reduced a dislocation after 24 days; Cooper and Stormeyer, after 42 days; Hutchinson, 60 days; Demarguay, 87 days; Donovan, 98 days; Milton, 114 days; Pollock, 120 days; Nichor and Gosselin, 130 days; Golding, 136 days, and McGraw[4] corrected a bilateral dislocation of four months' standing. Our patient had the double dislocation of almost five months' standing at the time of the operation.

Case 3.—M. R., white, female, aged 33, came to me with the following entrance complaint: Inability to close the mouth. In September, 1915, while yawning, the patient found that the lower jaw became immobile in the open position, and it had remained so since. It was painful at first and continued so for a few days. Since then there had been no pain, but the patient had not been able to close the mouth. Naturally, the patient could not bite or chew. Attempts were made to replace the

3. Brophy's Oral Surgery, p. 423.
4. Brophy's Oral Surgery, p. 424.

dislocation before the patient came under our care, but were unsuccessful. She presented the typical facies of a bilateral dislocation of the mandible. The inferior incisors protruded 1 cm. beyond the superior.

The patient was able to open the mouth 3 cm. Anterior to the ear was a depression formerly occupied by the condyle. The condyles could not be felt, as they had slipped forward underneath the zygoma in the zygomatic fossae. There was malocclusion of the third degree, and no deviation to the right or left.

Operation.—On Jan. 31, 1916, resection of the bilateral dislocation of the mandible by incision and local manipulation was performed under ether anesthesia, with a preliminary injection of morphin. Under deep anesthesia, an effort was made to replace the dislocation by placing between the molar teeth a rubber wedge containing an iron core, and exerting pressure on the symphysis upward. This was not successful. The condyles were not moved in the least.

On the right side, over the zygoma, a horizontal incision was made through the skin and fascia down to the bone, the incision being posterior and extending slightly upward. The length of the incision was two inches. The zygoma was separated from the malar bone anteriorly. The posterior incision through the zygoma was made as far back as possible, using a Gigli saw.

An attempt was made to leave the zygoma attached to the masseter muscle, when it was turned down, but in this instance the piece of bone fell off on the floor and was lost. The masseter muscle was turned down, and the condyle exposed in the zygomatic fossa. Using a wedge and the base of the skull as a fulcrum, the point of the wedge was applied to the condyle and pressure was exerted on the symphysis, the condyle being thus forced back into position.

The same operation was performed on the left side, except in this instance the zygoma was not lost, but remained attached to the masseter muscle.

When the left condyle was forced back into position the right came out of the socket. A second wedge was used to hold the left condyle in the socket, and then following out the procedure as outlined above, the right condyle was again forced back into the socket.

On the left side, the piece of zygoma was placed in its proper position, sutures being made through the periosteum of the zygoma and the neighboring bone. On the right side, the

masseter muscle was stretched back in position, and the wound closed with silk worm gut. During this time the mandible was held in position by firm pressure. A Barton bandage was now applied.

The patient made a nice recovery, experiencing no harmful effects from the operation or anesthetic.

Salines and brandy were administered per rectum immediately following the operation. Six hours later, water was administered through the nose by catheter. The patient was fed in this manner until the Barton bandage was removed. Four days after the operation the stitches were removed. Fourteen days after the operation the Barton bandage was laid aside. At this time the patient had good motion of the jaws. The occlusion was perfect, at least it was as good as before the dislocation, and the patient could open her jaws 3 cm.

The patient went home on Feb. 25, 1916, that is, about three and one-half weeks following the operation. At this time she could open her mouth to a distance of 4 cm., and mastication was excellent.

So far as we are able to determine, the method of replacing the condyle and using the wedge is original.

DISCUSSION

Dr. Carmody, Denver: One of the cases mentioned is very much like one of mine. I had a case recently in which the ankylosis was caused by actinomycosis. The history of this case is interesting, because this patient received about 67 ounces of 5 per cent. iodin to cure the actinomycosis, and then required an operation. The essayist speaks of removing the condyle. I have found in some cases this is not necessary. I had one case which has gone now about six years. I went through the neck of the condyle, removing about one-fourth inch of bone, placing fascia in between. In the case of actinomycosis, I removed the condyle. I wish to thank Dr. Boiler for bringing the subject before this society, as I believe it comes within our field.

Dr. Joseph Beck, Chicago: The last point that the essayist made is the point I want to emphasize, i. e., that this is borderland surgery and perhaps is not interesting to most of the members. Most of the cases originate from otitis media or mastoid disease in children, the necrotic process passing along the zygoma and infecting the mandibular joint, thus producing ankylosis. Here is a patient (illustrating photograph) who had an operation for this bilateral condition acquired in infancy. In the attempt to correct it an operation through the most dangerous part of the anatomy was done by a competent man. You see an incision made in the region of the parotid. The facial nerve paralysis and the fistula that formed in this case were no doubt distressing. The operation was done over by us in a classic manner as first suggested by Dr. J. B. Murphy. Here is another case (illustrating photograph) of a tumor in this region. A tumor had been removed by a general surgeon, and secondary to this the patient got an ankylosis of the temporomandibular joint. It was

impossible to do any operation by utilizing the temporal fascia, so I went from below; lifted up the masseter muscles, and with an electric burr made a sort of a condyle false joint, planting in between these two fragments a piece of neighboring masseter muscle. That gave the patient a pretty fair motion subsequently. I think the point in this paper is that every time one has a case of otitis media or mastoid with perforation in the temporozygomatic region, one should be on the lookout for subsequent ankylosis of the temporomandibular joint.

PROBLEMS OF THE DEAF

M. A. Goldstein, M.D.
ST. LOUIS

At the suggestion of the chairman of this section, and with the consent of the council and the program committee, we have arranged a practical demonstration of the work in lip-reading and normal articulate speech as we teach at the Central Institute for the Deaf, St. Louis. We do not assert there is anything radically new in the system of instruction that we are developing and advocating. We are closely watching the evolution of these educational problems and are taking advantage of every opportunity that presents itself to add new features and new ideas to strengthen pure oral training.

The system of teaching now being developed in our institution is the result of daily practical cooperative work of the trained teacher of the deaf and the experienced ear specialist. I think it is the first time that these forces have ever worked together in such a manner and to such a degree.

There are two systems of education of the deaf which claim your attention; one, the system dependent largely on signs and the manual alphabet; the other, pure oralism, whose advocates desire it to dispense with signs and manuals altogether.

The main argument advanced by the signers and combined system advocates against the oralists is, that the quality of speech heretofore obtained by oral methods is so unintelligible and so inadequate that it is not comprehensible to the average ear familiar only with ordinary speech. This contention is a fair one and I am willing to agree with the assertion of the signers that if nearly normal and intelligible speech is impossible by oral training, we should resort to the pad and pencil, the finger alphabet and signs. The principle, however, on which we have based our work in the development of the Central Institute for the Deaf depends on producing with each child speech of such quality, fluency, and volume that the normal ear can follow it intelligently and practically. If we can put our children in a position in which they can be emancipated from the class to which they have been consigned because of the loss of this important sense organ, I think the energies and the efforts, the

skill and the science, the experience and the patience, that have been devoted to the development of pure oralism are worthy of serious consideration and further study.

We have brought a small class of our pupils to this meeting for practical demonstration. One of our Fellows, who happened to be present when a similar demonstration was given before this Academy in Chicago last year, remarked: "We had this demonstration last year." True, we did, but it is only by constantly and persistently presenting our work to your attention that we can eventually hope to get your cooperation. We must have the assistance of the medical profession in order to spread our mission throughout the land.

Let me briefly give the status of these several pupils:

This little fellow in front of me is 6 years of age, was born deaf, and has had one year's work at the Central Institute.

The little girl with the pink ribbon in her hair is 10, was born deaf, and there is no caloric, galvanic, or rotary reaction with the nystagmus test.

Molie is the first product of the oral system as we have developed it. She has had two years' work with us. She is 10 years of age. When 3 years old she had cerebrospinal meningitis and lost her hearing completely. She is deaf by all functional tests. She is a normal child in every respect, excepting her hearing organs.

The little girl with black hair, dark eyes, and a white ribbon is 9 years of age; was born deaf, and came to us two years ago. She had a slight remnant of hearing at that time and could, with some difficulty, distinguish the vowel sound ah! from oh! We are not only educating this child by oral methods, but she is also a good example of reclamation of hearing. This little girl, as I will demonstrate, is beginning to acquire speech through the normal aural channels. She is beginning to repeat and carry on a conversation in sentences to the limit of her actual education.

The little kiddie in the back row has been in our school for only six weeks. We will demonstrate with her the first steps in training the deaf child. This child has never heard speech. Speech is only an imitation. We could not speak without hearing, and we are taught to imitate what we hear.

These children are taught to watch the mouth, the lips, the tongue, and all of the other accessory organs of expressed speech; are given proper breathing exercises, and take note

of all incidental conditions which are essential to the production of normal speech. We are carefully studying also the psychologic aspect of each pupil. We are developing every possible resource in the study of this important question. We are still experimenting and are gradually evolving a system of oral education that marks distinctly an advance of the work thus far accomplished. Sense-training is one of the keynotes in our daily practice. The touch sense of our pupils is unusually well developed. Their sense of rhythm is remarkable and becomes a splendid accessory in the development of speech.

We will endeavor to present our practical work in graded form, beginning with the first steps taken to interest the totally deaf young child who has never heard a sound, and will lead up, step by step, to the possible accomplishments of pure oral training.

Our demonstration will include also lip-reading by adults who have lost their hearing, and who have been trained to such a readiness that they can carry on their practical intercourse with the social world around them without serious handicap.

This demonstration will be conducted by Miss Hilliard, the principal of the Central Institute, and Miss Avondino, the senior instructor.

DISCUSSION

Dr. H. B. Young, Burlington, Iowa: As I have tried previously to make plain, I have great praise for the results shown by Dr. Goldstein, and as great condemnation of the conclusions which he draws from them. I have shown the fallacy in his reasoning in two communications to this Academy (to be found in the last two volumes of the Transactions), and it is therefore unnecessary to add anything at this time. However, instead of speaking myself, I desire the courtesy of the Academy for one better qualified to speak on this subject—in fact, two, Dr. J. H. Cloud of St. Louis, principal Gallaudet Day School for the Deaf, and the personal representative of the president of the National Association of the Deaf; and Mr. I. B. Gardner, superintendent of the State School for the Deaf at Little Rock, Ark. Dr. Cloud is himself a deaf man, a good speaker, but poor lip reader—a not unusual incident of oralism.

Dr. James H. Cloud, St. Louis: I am no orator like Dr. Goldstein, or Dr. Young, otherwise I should enjoy public speaking much more, but I greatly appreciate the opportunity to address you as a representative of the National Association of the Deaf, an organization which has over 1,500 members in all parts of the country. The membership is composed of graduates of schools of all methods of teaching the deaf, including the manual, the combined, and the oral. I am speaking to you this afternoon not as a little child whose education is beginning, but as one who has had experience in the world as a deaf man for over forty-five years. The deaf child of today is the deaf citizen of tomorrow. The citizen has the right to say by what method the child should be educated. The educated deaf of this country, in fact, of all countries, are a unit in endorsing all improved methods of teaching the deaf. If there is anything worth while

in the methods of teaching the deaf, the educated deaf are the first to appreciate the fact, but if there is anything in the system of educating the deaf which is deficient, and which they therefore do not approve, they are quick to say so. The combined system includes approximately more than 10,000 deaf children in the schools of the United States today. This system employs, writing, spelling, the sign language, and whatever else will be an aid in the education of the deaf. It does not confine the child to any single method, but adapts the method to the needs of the individual child. Such children as are able to make better progress by the oral method are taught by the oral method; otherwise, they are taught by the combined method, or by some other method which will give them the best possible education in the shortest time. The pure oral system depends on speech and lip reading exclusively, with writing included, so far as communication between teacher and pupil is concerned. With the oralists speech and lip reading are of paramount importance; education is secondary. With the deaf, education is of first importance. It is an open question whether the time given to teaching the deaf exclusively by the oral method is worth while in the long run. In the case of about 30 per cent., it is just about worth while; in the case of 60 or 70 per cent., the game is not worth the candle. It is time wasted. The orally taught deaf can acquire speech in the schoolroom, can learn to read the lips of the teacher, but after going out into the world and into business their lip reading is sometimes of very little use. In important matters it is unreliable. Such is the testimony of the orally taught as well as those taught by other methods. And the tendency among the deaf who have been taught by the oral method is to "backslide" into the sign language after they leave school, no matter what method they used in the schoolroom. Another thing—there is no orally taught person in this or any other country who can follow the proceedings of a convention like this. The sign language is equal to anything. It can follow any speaker, or any subject, and give the educated deaf the full substance of the discourse within the same time of its delivery. It is able to follow Billy Sunday, and if there is a lip reader who can do that I should like to meet him. It is often claimed that Germany is 100 per cent. pure oral. That is easily explained. The oral system is forced on the schools. In a country like Germany oralism is like compulsory service in the army. The schools can use no other method but the one required of them. It is only necessary to get to the ear of the minister of education concerning the method to be employed and the thing is done. In this country it is different. If the oral method needs to be boosted by legislation that amounts to a confession that it has not sufficient merit in itself to stand on. Why is it that the oralists go before school boards, state legislatures, and other law-making bodies and plead for the compulsory use of their method? The advocates of the combined system have done nothing of the kind. They are satisfied to let merit decide that matter. There is a college for the deaf in Washington—Gallaudet College—supported by the national government. It has been in existence over fifty years. It has received graduates from all the leading schools in this country and from some schools in foreign lands. It is a matter of record that graduates of combined system schools, as a rule, meet with less trouble in the entrance examination and after admission, than students from the oral schools. Such facts speak for themselves. The educated deaf of the land do not wish a body like this to commit itself to any one method without hearing both sides. The demonstration this morning was purely oral. Had we known that it would be a fair demonstration, one which would show all methods of instruction on their merits, there would be no difference of opinion, and no representative of the National Association would have

come here, but the fact that the oral propagandists are busy trying to influence certain bodies to their way of thinking makes it necessary for us to meet them on their own ground and to combat their influences. The president of the college at Washington, a few years ago, visited Germany, where 100 per cent oralism is the rule. He visited the school in the city where the founder of oralism used to live. He attended a lecture in the school chapel. He observed that the teacher in addressing the pupils used the sign language and spoken language at the same time. On asking the reason for the simultaneous use of speech and signs, he was told that speech was used to conform with the law and signs were used that the deaf might understand. There is a very good lip reader living in Kansas City. In her younger days she was educated by the oralists and much put forward by them. She has been out in the world a good long while and does not find lip reading what it was accounted to be. Some time ago she published an article in the *Oralist's Magazine* advocating that the public be taught how to talk with the deaf. As a lip reader she cannot get along very well with the public. She could understand her father, mother, and teacher. The trouble is, parents die and teachers leave. Not everybody looks at us when addressing us. Not everybody knows how to talk with the deaf. The sign language does not interfere with speech, and it is a fact that the best lip readers, the best educated deaf, those who can speak well, are also past masters of the sign language. The sign language is one of the greatest aids in the profession, and the source of great pleasure and profit to the deaf. It has been in use for over 100 years in the United States. Ten thousand children are in combined system schools today, while only 2,000 are in the oral schools. Any person who will visit the class rooms will observe they use a sign language in oral schools. This morning Dr. Goldstein made a distinction. He said there were "signists" and oralists. There is no such animal as a "signist.' There is a combined system, but there is no "signist." The combined system uses all approved methods while oralists insist on the deaf being taught by their one method. Dr. Gillett, a former president of the Oral Association in this country, told me time and again that when birds no longer fly, when fish no longer swim, then the deaf will stop using the sign language.

Miss Ethel M. Hilliard, St. Louis: This discussion takes me so by surprise that I hardly know what to say. It seems to me a very sad predicament, almost as bad, in fact, as would be a fight between doctors who, whatever their opinions on certain disputed points, are devoting their lives to raising the standards of living, to bettering conditions of health. We teachers of the deaf are devoting our lives to the cause of reducing to a minimum the handicaps under which deaf people must spend their existence. Whether we teach by signs, by manual spelling, or by lip reading and speech, our final object is the same. Certainly there should be no dispute among us, and it is not my intention to enter this discussion with the slightest feeling of bitterness. I do, however, wish to point out a few seeming fallacies in the arguments presented by the advocates of the combined system of teaching the deaf, which, as you know, incorporates the use of manual spelling and the signs in the education of deaf children.

Dr. Cloud, in the course of his remarks, said that the educated deaf are a unit in regard to their attitude on this mooted question; that the educated deaf unanimously believed in the combined system. He is leaving out of the count a great many people like Miss Kappler, whom you saw this morning. He claims that the deaf, no matter how educated, will revert sooner or later to the use of the sign language. Is not the

little lady who stood before you this morning reading my lips, to which she is acustomed, and your lips, which she has never seen before, a direct refutation of that? Can you imagine her with her splendid power of hearing, with her eyes, and speaking with her natural organs of articulation, preferring the sign languge to the ordinary means of communication? And she is only one of a large class of educated deaf people who know nothing about signs or manual spelling and who move comfortably in the social circles to which they normally belong.

In reference to the argument that no lip reader has ever been so perfectly trained as to be able to understand a discourse, it must be admitted that this is true. But suppose that the trained lip reader were able to understand even 10 per cent. of what was going on. Would it not be just 10 per cent. more than could be understood by the deaf person who had only signs at his disposal? It is true that, with an interpreter, the deaf person might get a great deal more, but the percentage of hearing persons thoroughly conversant with the sign language is very small, and it is not always convenient for the deaf person to have at his side a handsome young woman to interpret each word that is said. Listening to discourses, after all, occupies an exceedingly small part of even a hearing person's life. The beauty of lip reading was easily demonstrated to many of you this morning in the lobby of the hotel, where Miss Kappler conversed freely with any number of you with the greatest ease. A deaf person, manually taught, would have had to use an interpreter or resort to writing, which is admittedly laborious.

Dr. Cloud says, moreover, that people ordinarily do not know how to talk with the deaf so that their lips can be read. This remark might have been pertinent twenty-five years ago, when it was still thought that a certain amount of mouthing was necessary, or at least helpful. Today we do not teach lip reading that way. Naturalness of speech is the keynote of progressive educators of the deaf at the present time. Graduates of oral schools are being turned out every year who converse with any one and who hold certain positions simply because they are conversant with lip reading and can use speech.

Apologizing for personal references, I wish to put one question to you in a very personal way. Suppose this matter came close to you, as it is being presented to many parents today. Suppose there were born into your own home a child who had not the power of hearing and hence could not acquire speech except through special education. Would you, holding in mind the possibility of lessening the child's handicap as much as possible and being desirous of obtaining the best advice for your guidance in the training of this child—would you go for counsel to an educator of the deaf who had lost his hearing after having acquired speech and yet who had never taken the trouble to learn lip reading on his own acount? There is much opposition to pure oralism among deaf people, but it is usually confined to those who have been educated, at least in part, through signs, and who have not enjoyed the benefits of speech and lip reading in their own lives.

There is also the stock argument that pure oralists cultivate speech and lip reading to the neglect of general education. One little 10-year-old girl, whom you saw this morning, is doing regular fourth-grade work. Another, who came to us two years ago at 7, is doing second-grade work, both following the curriculum of the St. Louis public schools. Orally taught deaf children are ahead instead of behind children in the combined schools. It is true that we spend much time teaching lip reading and developing speech, but is it not perfectly plain that we cannot talk and read lips without something to talk and read lips about? Information, culture, general education go hand in hand with oral instruction.

Before I close my remarks I wish to make one admission. The criticism of the speech which is usually taught to the deaf is in many cases a perfectly just one, and I agree with the critics of oralism that speech which cannot be understood is valueless. Oralism in many of our state schools, working under the handicap of poorly trained teachers and lacking the support of the deaf and the authorities of the school, has too often been an argument against itself. But I submit that while the combined system has often been content with methods which belong generations back in the evolution of education, oralism has been forging ahead. It is at last coming into its own, and its present struggle is to throw off the shackles of antiquated methods and unreasoning opposition so that it may go forward even faster.

Sifted down to its real essentials, the controversy between the pure oralists and the advocates of the combined system is found to be one of proportion only. All are agreed that a certain percentage of deaf children can be taught by pure oral methods. This number is thought by one side to be exceedingly small and to consist of a few exceptional individuals. We oralists believe that the vast majority of the deaf stand in no need whatever of any other language than English, their mother tongue, which, in spoken language taught mechanically and understood through the eyes instead of the ears, can be used comfortably as a medium of communication; and we feel very strongly that, till every deaf child has been given the opportunity to prove fairly whether or not the oral method is adapted to his needs, our duty to the deaf has not been done.

Miss Avondino, St. Louis: I can give no better example of the poor results in speech, as well as in mental development, in combined schools than a young girl I brought to the Central Institute with me from Oklahoma. I taught this little girl six years ago. Then she was in the same grade as Mildred, which is the latter half of first year work. In October, when our children were assigned to their classes, this young girl, now 17, was unable to go in the grade with a 9-year-old child who has been in school only two years. Having taught both, I feel that the slight difference in mentality would not warrant such a difference in their development. But I do think that her educational progress was hampered by the use of signs.

The acquisiton of any language requires constant practice, and a working knowledge cannot be obtained in the college courses commonly given. If this is true of people with normal hearing, it is evident that it is utterly impossible for the deaf to learn speech in the time devoted to it in combined schools. The line of least resistance is what a child is going to take. We all do the easiest thing, whether it is better for us or not, and that is where the danger comes in. Once a child acquires the attitude of not wanting to talk, it is extremely difficult to instruct him in speech and lip reading against this indifference. I have taught nine years in combined schools, and my results in speech with young children were just as good as in a pure oral school, but as speech and lip reading received absolutely no encouragement outside of school hours, these results were practically nullified. Teaching speech is difficult even under favorable conditions, and the poor results obtained in schools where signs are allowed to interfere are only to be expected.

The great cry by advocates of the combined system is that lectures cannot be followed through lip reading. I know many hearing persons who never miss a lecture, but whose happiness is not one bit greater than many others whom I know who read lectures from current periodicals. Not being able to understand a lecture in no way discounts the value of being able to carry on an ordinary social or business conversation.

I know a very capable man who, on account of his inability to read lips, was compelled to take an inferior position, though at the time there were three good positions available, any one of which he could have filled creditably if he had been only able to understand the conversation pertaining to that particular line of business.

The impression that mental development is not as great when children are taught by means of speech as when signs are used, has been proven erroneous by a comparison of children taught by both methods. In fact, it is conceded by the majority of educators of the deaf that the mental development is greater under pure oral instruction. This little boy is only 7 years old, and his understanding of both spoken and written language is above the average 7-year-old deaf child. In our school we have a number of children who were considered slow in other schools, and their progress has been as rapid, in comparison, as that of the brighter children.

Dr. M. A. Goldstein (closing discussion): In advocating pure oral training for the deaf and in presenting the question of the respective merits or inefficiencies of the several methods of education of the deaf for a strictly scientific consideration, it has always been our purpose and policy to meet every issue squarely and equitably. In closing this discussion I propose to state a few plain facts.

About three years ago the superintendent of instruction of the public schools in St. Louis requested me to make a careful functional test of every pupil in the Gallaudet School, the public day school for the deaf in our city. The detailed report of the result of this examination has never been presented by me to the superintendent of instruction. The spirit of defiance and criticism with which the principal of the Gallaudet School, and some of his supporters, have attacked me here and elsewhere warrants the inference that I found nothing in the system of education prevailing in the Gallaudet School subject to unfavorable comment. The trend of events forces me to publicly present my observations now.

I found one little girl of 6 years of age in whom a radical mastoid exenteration had almost destroyed the hearing of one ear. The other ear was *normal*. This child was in the kindergarten class. The teacher was a deaf woman who communicated with the children by signs only. The children were signers. The little girl referred to had normal speech, but was compelled to continue her kindergarten work in a class of deaf children who were instructed entirely by signs and by a teacher who was totally deaf. What will become of this child's fluent and acquired speech? To place such a child in such a class and under supervision of such a teacher is an educational crime.

I found a number of other children who could hear the loudly spoken voice at a distance of from two to five feet from the teacher. These children could be segregated in small classes with a hearing teacher, and could obtain their instruction as normal children may. It would simply require more individual and special attention on the part of the teacher and the children to be trained in groups of two, three, or four. Instead of that, these children are taught by the manual, by signs, and lip reading. Their speech is being neglected and the natural avenue of education directly through the ear entirely disregarded. This is another educational crime.

The Gallaudet Day School for the Deaf of St. Louis, a public institution, is under the supervision and instruction of Mr. J. H. Cloud, a totally deaf man. It is this principal who shapes the policy of the public day school for the deaf of St. Louis; a man who, because of his inability to hear, is not qualified to judge whether a child taught orally has the proper quality, quantity, or inflection of voice; who, because of his defective hearing, is handicapped in his judgment as to the respective

merits of the several systems of education of the deaf. I am unable to explain why St. Louis, a city which enjoys the reputation of one of the finest public school systems in the world, should still cling so tenaciously to antiquated methods of teaching the deaf, when such cities as New York, Chicago, Philadelphia, and nearly all other metropolitan communities in this country, have recognized and established oral schools for the training of their deaf charges. Even the state school of Pennsylvania, at Mount Airy, under the direction of Dr. Crouter, than whom there is no more able representative among the well known authorities in the training of the deaf in this country, has established a pure oral system of training. The signs of educational progress are evident everywhere, even in the various state schools for the deaf. Here the oral sections are gaining in numerical and pedagogic strength yearly.

There is a tremendous scarcity of good oral teachers. Politics, nepotism, lack of funds, and other unfavorable influences, have done much to retard the growth and development of pure oralism as a modern and progressive method of education of the deaf. But oralism stands on a firm foundation. If our Fellows of the otological and medical profession would lend their support more definitely, and would assume their obligations and responsibilities more earnestly in the work of promoting the interests and educational opportunities of the deaf, this splendid mission would receive a more rapid impetus throughout the land.

I have been studying these problems seriously for nearly a quarter of a century. You can count on the fingers of one hand the otologists of America who have practically championed the cause of the deaf or given this subject much attention. A few of us who have engaged in this special field have received encouragement in words only: "It is good work, old man, keep it up." You cannot expect work of such importance, work that carries with it so much responsibility in every community, to be shouldered by a few men; you must *all* put *your* shoulders to the wheel. We still have much resistance to overcome, especially in the antagonism of the advocates of antiquated methods. We need the united support of an energetic medical profession in order to maintain the assertions which pure oralism has made.

The superintendent of the state school of Arkansas tells you, "you are nothing but laymen." You may be laymen from his point of view, but you are members of an influential and dignified profession that has as much responsibility to the community for the proper care and education of the deaf as has the honorable superintendent of any state school for the deaf and his corps of teachers.

I venture to say that very few of you have ever visited an institution for the deaf and carefully investigated the system of instruction used in such institution, or have sat through a demonstration by pupils and teachers of such institution, or have drawn your inferences from such a demonstration with a knowledge and experience that your profession has qualified you to exercise. If you have not done this you have shirked your obligations. The community is obligated to offer the deaf child the best education, just as it is obligated to offer the normal child the best education. You are an important factor in shaping the opinion of the community. There is so much that you can do in the study of the deaf child; in advising the parent and the teacher concerning the proper hygiene and care of the deaf child; in informing the community of the baneful and serious results of marked adenoid vegetations, hypertrophied tonsils, long continued suppuration in the ear; the prompt attention to the nose and throat in the eruptive fevers, so as to avoid ear complications; the question of consanguinity in marriage; careful functional hearing tests for each child so as to indicate his individual status in the schoolroom.

There are innumerable directions in which your energies may be applied for the advancement of the deaf.

True, it is financially an unremunerative question to the profession, and I am ashamed to admit that perhaps this may be one of the reasons why more members of our profession have not more actively interested themselves in it.

This absurd controversy between the signers and combined-system advocates on the one hand, and the champions of pure oralism on the other will soon adjust itself. All education is an evolution, and the evolution of the education of the deaf has pure oralism for its goal.

The superintendent of the State School for the Deaf at Arkansas states that we have 56 per cent. oral instruction in this country at present; thirty years ago we had 20 per cent., and as soon as we can secure more properly qualified and well trained oral teachers we shall have 75 per cent.

Germany has shown us an efficient way in the education of the deaf with 100 per cent. oral training. Politzer, Bezold, Urbantschitsch, and other German otologists of good, ripe experience are men who are just as preoccupied professionally as you are, but they have found much time to study the work of the education of the deaf. France enumerates over 70 per cent of oral training; Sweden and Norway over 75 per cent. Compare these evidences of oral development abroad with the 56 per cent. of oral training in America, and you will note that America has not yet proved her right to be recognized as a nation in which efficiency in such special education has reached its high point.

FRONTAL SINUSITIS AS A PROBABLE CAUSE OF ACUTE NEPHRITIS

GEORGE F. KEIPER, M.D.
LAFAYETTE, IND.

Last year before the joint meeting of the Middle and Western Sections of the American Laryngological, Rhinological, and Otological Society, in a paper entitled "Mastoiditis, a Probable Cause of Acute Nephritis," I reported two cases in which the mastoiditis in each instance was at least an accompaniment of the co-existent acute nephritis, if not its most probable cause.

As stated in that paper, the literature upon the subject is quite barren. Up to that time only two reports had been made, one by Dr. Joseph A. White of Richmond, Va., in 1904, entitled "Report of a Case of Mastoiditis, Complicated by Nephritis and Erysipelas, with Remarks," and one by Dr. Thomas Hubbard in 1908, on "Diabetes and Bright's Disease in Relation to Suppurative Osteomyelitis of the Mastoid." Both papers were read before the different sessions of the American Laryngological, Rhinological, and Otological Society.

In the discussion that followed the reading of my own paper, Dr. Derrick T. Vail of Cincinnati reported an additional case. To date these are all the reported cases.

As a supplement to my own paper of March 2, 1915, I now report a case of *purulent frontal sinusitis*, showing co-existent acute nephritis.

CASE 1.—Chester H., aged 15, of Swanington, Ind., was referred to me Jan. 11, 1916, by his physician, Dr. George R. Clayton, Jr., of Fowler, Ind. I saw the patient immediately after his arrival at St. Elizabeth Hospital of this city. His history is briefly as follows: About a year ago his forehead became very tender over the left eye and he was very sick. Under the treatment pursued at that time, the patient made a quick recovery; afterward, as his father stated, "the nose broke and run a large amount of matter." This was evidently a frontal sinusitis. Thereafter he appeared perfectly well until his last illness.

For two weeks prior to his admission to the hospital, the right side of his forehead had been very tender to touch and pressure. Treatment had been of no avail, and at last, yielding to the importunities of his family physician, he was brought

to Lafayette. His forehead over the right eye was still very tender to touch and pressure. His mind was confused, and at times he manifested mild delirium. His pulse was 124 and his temperature was 103.2 F. He was taken to the Roentgen-ray laboratory and skiagraphs of the frontal sinuses were made. These were negative.

A blood count was made, with the following result:

Hemoglobin	90 per cent.
Red blood cells	4,400,000
White blood cells	18,400
Differential count:	Per cent.
Polynuclears	86
Small lymphocytes	12
Large lymphocytes	2
Eosinophils	0
Mast cells	0

Examination of his urine was as follows: Color, dark amber; specific gravity, 1.020; reaction, acid; albumen, present; sugar, absent.

The microscope showed: Few epithelial cells; few white blood cells; amorphous urates; calcium oxalate crystals; few granular casts.

For the general physical examination, Dr. Frank B. Thompson of this city was the consultant. He did not find any lesion of the heart, lungs, or blood vessels.

Examination of his nose found it free from pus. His throat was normal. This was at 2 o'clock in the afternoon of the day of his admission to the hospital.

At 4:30 p. m. his pulse was 140 and his temperature was 104.2.

At 7:30 p. m. his pulse was 130 and his temperature was 102.2.

At 10:30 p. m. his pulse was 144 and his temperature was 103.4.

January 12:

At 3 a. m. his pulse was 140 and his temperature was 102.

At 7 a. m. his pulse was 142 and his temperature was 102.

At 8 o'clock I opened the right frontal sinus externally. It was full of pus, which was thoroughly cleaned out. No sinus could be found in the posterior table of the frontal bone. The frontonasal duct was occluded. This was opened and enlarged. His condition becoming bad, we gave the nose no further attention and hastily completed the operation and returned him to his room.

On recovery from the anesthetic, he became very noisy. Morphin sulphate was given him in a quarter-grain dose to quiet him. This was at 10:15 a. m. Respiration became rapid and the pulse weak. Camphor oil was administered subcutaneously in a 30 drop dose. This accomplished nothing. The

delirium deepened for a while; then he gradually failed and died at 3:45 p. m. the same day. No autopsy was permitted.

The pus obtained during the operation was inadvertently destroyed, and hence it is with regret that we write that no examination for its bacterial content was possible.

I have searched the literature in vain for a parallel case. It is barely possible that many more such cases exist. If care is used in making a thorough physical examination, as well as a local one, this complication may be more frequently observed, and we shall be in a better position to declare that purulent frontal sinusitis may be the cause of acute nephritis. In fact, every similar case that comes before us should have all the advantages offered by a complete physical examination.

ACUTE OTITIS MEDIA OF DIPHTHERITIC TYPE

Wm. C. Bane, M.D.
DENVER, COLO.

The literature on diphtheria of the ear is not voluminous, and most of the articles on the subject were written within the past twenty-five years. In 1889 Billington referred to diphtheria of the ear, and remarked that the exciting organism was undiscovered. In 1905, Welch and Schamberg mentioned merely that the disease is due to extension of the exudate from the pharyngeal vault. Ballenger, in his book, stated "that infection of the external auditory meatus is seen in rare instances in which there is diphtheritic otitis media with extension through the tympanic membrane"; and also that "otitis media as a complication of diphtheria occurs in only 4 to 6 per cent of the cases." Borden found the percentage much lower than this. Barnhill and Wales divided otitis media caused by the Klebs-Löffler bacillus into three classes, namely: (1) Primary diphtheritic inflammation of the middle ear. This is very rare. (2) Diphtheritic otitis media by extension from the naso-pharynx. There are a few cases of this type. (3) The most common form is the secondary middle ear infection, which accompanies diphtheria of the throat. Lewin stated that acute otitis media often accompanies genuine throat diphtheria. Phillips expressed the opinion that primary diphtheria of the ear is very rare, but that otitis media purulenta acuta occurs in 10 per cent. of all cases (Duel), and that it shows a marked tendency to become chronic.

I have reviewed the literature on the subject rather carefully, and the above brief references are typical, I believe, of most of the articles referring to diphtheria of the ear. They are meagre, and reference is usually made to otitis media accompanying throat diphtheria, rather than to real infection of the ear with the Klebs-Löffler bacillus.

Symptoms and Course.—Phillips also stated that the disease usually comes on at the height of the naso-pharyngeal process, and the infective bacteria enter the tympanic cavity through the Eustachian tube. Diphtheria bacilli, once in the ear, are likely to remain there for a long time, but lose much of their virulence after the subsidence of the acute symptoms.

The pain in the ear is severe, and persists for several days after drainage is established. Rupture of the drum-head is apt to occur early in the inflammation. The deposit of the diphtheritic membrane appears on the drum and in the auditory canal where the surface has been deprived of its protective epithelium. The membranous exudate has the characteristic dirty-white appearance and adheres firmly to the underlying tissues. The lymphatic glands back of the ear and in the cervical region become infiltrated and tender. The discharge from the ear is rather scant for the first few days, increasing as the membrane exfoliates. The mastoid and adjacent areas are often congested and painful.

Prognosis.—The tendency is for the disease to become chronic, for the perforation in the drum-head to persist, and for granulations to appear in the opening. Impairment of the hearing is marked, but improvement usually comes with the subsidence of the inflammation, although permanent damage may result.

Treatment.—This is constitutional and local. Antitoxin is imperative in the early stage, and is to be repeated according to indications. Locally, early free drainage is indicated, and the ear should be cleansed with hot boric or salt solution. The irrigation of ear, nose and pharynx with a 0.25 per cent. solution of formaldehyd several times daily will aid in bringing about a destruction of the diphtheria bacilli. This latter treatment was advocated in 1914 by H. R. Miller, of New York, and has proven very efficient in several cases.

The following case which I report, I believe to have been one of the few cases of true diphtheria of the ear. Dr. W. C. Mitchell, bacteriologist for the city of Denver for many years, can recall seeing but one case of diphtheria of the ear other than the one I now report:

Case 1.—W. A. P., aged 72 years, developed acute inflammation of the right ear Aug. 16, 1916. The following day the drumhead ruptured and he came under my care on that day. There was a free serous discharge. To make sure of ample drainage, I incised the drum under local anesthesia. On the day following the incision, the discharge was not free; the patient was suffering considerable pain, and on examination I could not make out the line of my incision, due to a grayish-white deposit on the lower half of the drumhead, and in the angle between the drumhead and the floor of the canal. I re-inserted the knife, and learned by the sensation of touch that the opening was free.

The patient was feeling badly, and suffering pain back of the ear. He was given 10 grains of aspirin every four hours. On the following day there was little improvement in the ear; no change in the appearance of the drumhead or canal, and only a slight discharge. There was some diminution of the pain. The exudate looked like a diphtheritic membrane. A culture was taken, which on the following morning was reported positive. The patient was then sent to the Steele Hospital for Contagious Diseases, given antitoxin, and placed under the care of Dr. Lingenfelter.

Cultures were taken from the nose and throat, and these cultures proved to be positive, although there appeared to be nothing in the nose or throat suggestive of diphtheria.

On entering the hospital, the patient's temperature was subnormal, pulse 88, respirations 20. The next day the temperature was 100.4, and following day 99.6, due no doubt to the reaction from the antitoxin. After several days of a subnormal temperature, the records were omitted.

The right ear continued to discharge moderately, and the patient improved in general health for about two months, during which time the cultures remained positive. I was then called in on account of a severe pain in the left ear, accompanied by some tenderness of the mastoid and darting pains throughout the left side of the head. I incised the drumhead of this ear, and it then began a course similar to that of the right ear, except for the absence of any membrane. The cultures remaining positive, I ordered irrigation of the nose, throat, and ears several times daily with a 0.25 per cent. solution of formaldehyd. Within four days the cultures were reported negative, and the patient was then dismissed from the hospital.

Since that time I have treated him frequently at the office. The left ear discharges very little, and what moisture does collect there, I believe, comes from the throat through the small perforation in the drumhead. The right ear has not done so well. The perforation remains about 2 by 4 mm. in size, with granulations showing through it, and the discharge, now due to mixed infection, is at times profuse. Various methods of treatment have been used in an effort to dry up the discharge, but without much benefit. The high frequency current seemed to make it worse. Chromic acid applied to the granulations was of some benefit.

I should not care to operate in this case, on account of the patient's advanced age and his weakened physical condition. He has also a chronic diseased condition of the left antrum of Highmore, which is being treated by irrigation. The duration of the otitis media has been prolonged beyond my expectations. However, it has been in keeping with the prognosis of these cases as given by those who have had experience with them.

BIBLIOGRAPHY

Billington: Book published in 1889.
Welch and Schamberg: Book published in 1905, p. 626.
Ballenger: Diseases of Nose, Throat and Ear, 1911, p. 482.
Barnhill and Wales: 1911 ed., p. 72.
Phillips: Diseases of Ear, Nose and Throat, 1911 ed., p. 451.
Borden, C. B. C.: Boston Med. and Surg. Jour., Feb. 13, 1913.
Miller, H. R.: Method of Treatment of Diphtheria Carriers, Med. Rec., New York, July 25, 1914.

DISCUSSION

Dr. John W. Murphy, Cincinnati: I have not treated a case of primary diphtheria of the ear. I think the statement given by one writer that from 4 to 6 per cent of middle ear infections are diphtheritic in character must be too high, as it has never been my misfortune to even see one cause that I considered due to this cause. The principal point of interest is whether the infection was primary or secondary to a throat trouble extending along the eustachian tube. The essayist states that while the mouth and the appearance of the pharynx surface were negative, the culture was positive, so I am inclined to believe that infection did occur along the eustachian tube. This patient had also a chronic empyema of the antrum of Highmore, and it is easily possible that the infection might have occurred from this source. I have seen several cases in which the membrane extended from the pharyngeal surface into the nasal cavity, and in one case both conjunctival sacs extended no doubt along the lachrymal ducts. Treatment of the case, I think, is all that could be expected. The fact that we are not on the lookout for these cases may cause us to overlook some of them, thinking that we are simply dealing with the acute purulent otitis media, we do not make a culture. Should cultures be made more frequently in these cases of discharging ears, I have ino doubt more cases like this one could be discovered.

Dr. Weil, New Orleans: I think we all agree with Dr. Bane that primary infection of the middle ear of this kind is extremely rare, so rare that I cannot quite conceive it would occur without getting into the ear through the eustachian tube, and I believe where the throat shows nothing in all those cases cultures will be positive from the throat; especially in view of the fact that we do have these cases of nasopharyngeal diphtheria, with a slight membrane in the nasopharynx and nothing in the pharynx or tonsils. I believe all of these cases are secondary, and, with Dr. Murphy, I believe if we made an examination oftener we should probably get a positive culture. I want to speak of the importance of the middle ear in the consideration of diphtheria carriers. It happened about four or five years ago that I had under observation some twenty-four chronic diphtheria carriers in whom I used vaccine. Three of those patients had chronic otitis. The discharge from the ear also showed the presence of the Klebs-Löffler bacillus, while a culture from the nose and throat did not, and in another case the throat and the nose showed the culture to be positive. The nose and throat cultures cleared up and were negative long before the ear.

Dr. F. R. Spencer, Boulder, Colo.: Unfortunately, I was called out and did not hear all the paper, but this paper emphasizes one point, viz: if we made a routine procedure of examining the discharge, we should make better diagnoses and the cases would not be so rare. They are not so rare as we might be led to expect. I know how thoroughly Dr. Bane does things. Too many of us, probably, do not do things as thoroughly as we should. If we did, our diagnoses would be better, and we should have a better classification of diseases.

Dr. Frank Vinsonhaler, Little Rock, Ark.: Last year at the Dallas meeting of the Southern Medical Association I reported two of these cases, one preceded by nasal diphtheria and an inflammation of the maxillary and frontal sinuses, both of which required operations. Later on, the middle ear became involved, and subsequently the mastoid. The first examination revealed only the influenza bacillus. If the diphtheria bacillus was present in the beginning it was not discovered by the bacteriologist, but when the middle ear and mastoid became involved the discharge showed the presence of diphtheria bacillus. The use of antitoxin in that case brought very prompet relief. The other case I reported happened to be my own. I was examined by my colleagues, who made the diagnosis of influenza. I developed an otitis media, and on examination it was decided not to incise for a few hours. In the meantime the middle ear ruptured. The membrane made its appearance in twelve hours after the rupture of the drum, and the discharge showed the presence of diphtheria bacillus. I was given 10,000 units of antitoxin, and it was very interesting to me to feel the influence of the antitoxin on the toxemia. My temperature fell and a general feeling of well-being came on in twelve to twenty-four hours. The purulent discharge ceased in a week. The ear healed with very little impairment of hearing.

Dr. D. D. McHenry, Oklahoma City: In one case the man had a great deal of pain following a general illness diagnosed la grippe, and reported several times that large masses of material had come from the ear. Two weeks after the discharge commenced, he came to me with a large perforation and profuse discharge. I made a slide of it, and it looked more like a Klebs-Löffler bacillus than an influenza bacillus. A culture was made which was positive. The sequel of the case was that several months were required in healing. He absolutely refused to have any toxin, and the general practitioner was not in favor of it. The ear finally healed, leaving a large perforation and very decided defective hearing.

Dr. J. M. Ingersoll, Cleveland: I had one case of diphtheria of the ear, a young girl with chronic suppuration. She had had diphtheria four months previously. She was admitted to the hospital, and cultures taken from the throat were negative. Cultures taken from the mastoid when the radical was done showed pure diphtheria culture. Then cultures were again made from the tonsils, and we found a diphtheria carrier. We did a tonsillectomy after the mastoid operation. The diphtheria bacillus in this case apparently had no influence on the healing process, either in the ear or in the tonsil.

Dr. Carmody, Denver: One point as to diphtheria carriers. One of our prominent oto-laryngologists, a few years ago, contracted scarlet fever doing a mastoid after the child was out of quarantine. So we can get infection from some of these discharging ears. As to antitoxin, I saw a case a few days since, and the membrane had entirely disappeared one and one-half hours after the use of antitoxin.

Dr. Bane, Denver (closing discussion): Possibly my case was a so-called diphtheria carrier and became manifest with the development of the otitis media. The discussion has revealed the fact that other cases of otitis media, with Klebs-Löffler bacillus infection, have existed, and might have been reported before this.

THE SAFEGUARD AGAINST THE RADICAL MASTOID OPERATION, THAT IS EARLY DRAINAGE THROUGH THE MASTOID ANTRUM

SECORD H. LARGE, M.D.
CLEVELAND, OHIO

The subject of this paper was suggested to the author by the death from meningitis and cerebral abscess of a young girl, 18 years of age. She had had a suppurating ear since childhood, following scarlet fever. She was taken care of by the family physician, who told the mother it was better to have the ear discharge, as that was a safeguard against brain complications. In a recent paper I advocated the opening of the mastoid antrum, after three weeks of conservative treatment, in those cases of uncomplicated suppurative otitis media that showed no improvement, and I was criticized by a number of my friends who thought this was too radical a procedure.

We as otologists must arrive at some definite conclusion, so as to go to the general practitioner and state emphatically that a discharging ear is a serious affection and should be drained through the mastoid antrum after a limited time of conservative treatment. If this could be accomplished, we should have very few chronic cases. There naturally will be a number of exceptions to a time limit. A bacteriologic examination is very important, especially in the acute stage, because in the chronic stage your culture generally shows a mixed infection. If a streptococcic infection, especially that of the streptococcus capsulatus, we should operate earlier on account of its rapid progress in invading vital structures. This is also true in cases of contagious diseases in which the middle ear is involved.

I sent out the following questions to a number of otologists:

1. In an uncomplicated case of acute suppurative otitis media, how long would you treat it conservatively before opening the mastoid antrum?

2. After opening the mastoid antrum, how long would you wait, provided the case showed no signs of improvement, before performing the radical operation?

3. In an uncomplicated case of suppurative otitis media, provided you had not had the care of the case during the acute

stage, how long would you treat the case conservatively before performing the radical operation?

Dr. Dunbar Roy, Atlanta, Ga.:

1. Would treat it conservatively for several weeks, if after the first week all the symptoms began to subside. The condition of the patient and the clinical symptoms must be our guide in this matter. Every case differs.

2. In this, too, there is no fixed rule as to time. There would be decided complications if the mastoid did not heal.

3. Every case differs. You can tell very readily if conservative treatment is having any effect, and if after a month there is no improvment, then put it up to the patient as to an operation.

Dr. George L. Richards, Fall River, Mass.:

1. Several weeks; perhaps two or three months. Depends on rate of discharge and whether stopping or not. If profuse and not diminishing, would operate after two weeks, or as soon as consent could be obtained.

2. Hard to answer dogmatically. Being by nature conservative, I should probably wait some time. Amount of discharge, degree of hearing, size of perforation would all influence. If perforation is high up in Shrapnell's membrane or Prussahs' space, it is less likely to heal than such large ones in inferior quadrants.

3. In our clinic and private practice, especially in children, we often treat these cases several months. May dry up and disappear, and then perhaps return months afterward. We go through the process once more, usually, and on the next return, if they come back, to the radical operation. Unfortunately, in my hands I often find a more or less prolonged after treatment necessary, even when the radical has been performed before a clear dry cavity remains. Possibly this is because I have never done skin grafting, so that I do not approach the radical operation with any too much enthusiasm as to rapid cessation of all discharge. I do feel, however, that the danger to the patient's life is obviated by the radical operation.

Dr. J. F. McKernon, New York City:

1. Four to six weeks. Much would depend on the physical signs presenting.

2. Would not perform it unless there was evidence of middle ear necrosis.

3. Would depend entirely on the physical signs presenting and the symptoms present.

Dr. J. W. Murphy, Cincinnati:

1. I would say from three to four weeks.

2. Not longer than six weeks, as these are the cases in which I consider delay dangerous.

3. If the symptoms were not urgent, I would wait some time, say eight to ten weeks, as with a fair degree of hearing in the ear, I should hesitate about doing a radical operation. These are cases that always cause me considerable anxiety to know just when to pursue the more radical course.

Dr. Phillip D. Kerrison, New York City:

1. I am unable to give any dogmatic answer to this question, because the factors on which the question of operating depends cannot well be reduced to writing. On a case with continued fever (septic absorption), tender-

ness marked and over an extended area, and profuse discharge, I might operate very promptly as a matter of safety. In another case, with exactly the same symptoms, but in less exaggerated degree or form, I might delay operation and bring the patient through eventually without surgical intervention. Commonly discharge and tenderness are the main symptoms, and so long as these are not too pronounced, and particularly if they show any tendency to improvement, I am inclined to wait.

2. After a mastoidectomy in a case of otitis media purulenta acuta, I have never had occasion to resort to a radical operation. If the case required secondary operation, and provided the lesion was not a chronic suppuration, a careful secondary mastoidectomy seems to me all that is called for. On the other hand, we have all, I suppose, had cases of chronic suppurative otitis media in which we have tried a modified mastoidectomy, in which later the radical operation has become necessary.

3. In a case of otitis media suppurativa chronica, previously under the care of another man, I am inclined to consider only the lesion, i. e., the symptoms, and await conditions as I find them, and try my own plan of treatment, irrespective of the other man's efforts and experience.

Dr. Samuel J. Kopetzky, New York City:

1. The indications for opening the mastoid antrum do not in my opinion rest on a point of time at all. In some cases the time is next day, in others never. Months and years may elapse and no indications be present to warrant operation.

2. The radical operation is not indicated as a secondary procedure to cure an unhealed acute mastoiditis. I cannot, therefore, give an intelligent answer to your question. Whenever bone necrosis is present, etc., the simple mastoidectomy is not sufficient, and should not be performed in the first place.

3. Again, I make no time limit. I first try to diagnose the lesion which produces what we clinically call suppurative otitis media, and treat it accordingly. If the lesion presented is one which requires radical operation, I do this at once; if it does not, I institute the treatment required. There is so much confusion in the use of terms that I hardly understand what you are driving at. For my part, chronicity is not a question of time; it is a question in pathology, and a given lesion requires a given line of treatment. The emphasis should be laid on the lesions which present themselves. These should be diagnosed as exactly as possible, and the therapy will then follow as a matter of course. If I am able to come to the meeting, I will try to make my point clearer.

Dr. Samuel Iglauer, Cincinnati:

1. About six weeks.

2. I should probably clear out the mastoid a second time before performing a radical. This would be three or four months.

3. I should treat the case until six weeks had elapsed from the time of perforation of tympanic membrane.

Remarks.—In all doubtful cases I should be guided by the tendency of the discharge to increase or diminish and should lay great stress on the roentgen findings.

Dr. John F. Barnhill, Indianapolis:

1. Time varies largely, due to character of infection. In strict cases, think operation often indicated in forty-eight to seventy-two hours. In milder cases, if drainage shows no tendency to subside after two weeks, operation is advisable, even though no serious symptoms are present.

2. I never have as yet advised a radical mastoid operation for an acute case. I can conceive of a necessity, but think it rare. If the acute case is not cured by simple mastoid operation, complication has arisen, or the operation was incomplete.

3. I presume you refer to chronic suppurative cases. If no danger symptoms arise or are present, three or even six months may be permitted in earnest effort to arrest the discharge. Success sometimes follows and the ears are in better sterile condition for operation, should it be ultimately necessary. If danger symptoms are present (headache, dizziness, fever, etc.), would operate at once.

Dr. J. E. Brown, Columbus, Ohio:

1. Ordinarily, should give approximately six weeks, unless an unusually profuse discharge was present, or continued infiltration of drum membrane indicated the mastoid operation to conserve hearing.

2. I could not give a time rule for this.

3. If I had treated the case for any period over two weeks without seeing any improvement during that time, I should feel that a radical mastoid would probably be required for a cure. I think a simple mastoid suffices to cure many of these cases, especially if the suppuration has existed less than six or eight months.

Dr. Clarence J. Blake, Boston:

1. As long as required to stop the discharge and effect cicatricial healing, unless the mastoid antrum became implicated beyond reach of the middle ear treatment.

2. Should prosecute treatment unless the mastoid cavity became implicated, and then operate with a view to enlargement of the canal access and prompt closure of the postaural wound.

3. According to the demands of the individual case. No time limit, therefore, determinable.

Remarks.—The arbitrary setting of a time limit for the treatment of a suppurative disease of the middle ear is, in my opinion, as much a mistake as the assumption of a chronological age. Each case must be treated on its merits, both local and systemic. The question of an invasion operation must be one of condition and not of time, and the question of acoustic value should be weighed as well as the lethal consideration for or against any operation more extensive than curettage, ossiculectomy, or direct opening of an invaded labyrinth.

Dr. J. A. Stucky, Lexington, Ky.:

1. If the discharge from the ear was very copious at the end of a week or ten days and the patient showed evidence of absorption of pus, I should open the antrum.

2. If the patient showed no signs of improvement thirty days after this operation, but, on the contrary, became more septic, and I felt confident I had removed all the mastoid cells when the first operation was performed, I should then do a radical operation. This operation, however, I consider the last resort, and I should not perform it until I had exhausted every other means of getting rid of the septic condition.

3. If a thorough classical mastoid operation had not been performed, I should do this, and then treat the case for several weeks. If they did not show improvement, but, on the other hand, steadily got worse, I should resort to the radical operation.

Dr. C. R. Holmes, Cincinnati:

1. Do not think any positive length of time can be given, as it must depend on symptoms and the amount of discharge. Slight discharge might be limited to the middle ear and profuse discharge means involvement of the antrum whether there is tenderness and fever or not, and I should open that, as a rule, after two weeks.

2. If it is an acute inflammation in the ear that was normal before, it should not come to a radical operation at all if operated on in time. I should not make a radical operation under three months.

3. Two or three months from the time of the primary onset of the disease, but I should first remove the adenoids and tonsils, and operate on the nose and sinuses with the hope that the radical operation might be avoided, as it is a serious matter to deprive a patient of so much hearing if it can possibly be saved.

Dr. J. M. Ingersoll, Cleveland:

1. A profuse purulent discharge, continuing unabated under careful treatment for more than three weeks, is almost certain evidence of involvement of the mastoid.

2. If a mastoid operation is done within one or two months after the beginning of an acute otitis and fails to heal, I should think that some foci of infection were left at the first operation, and should endeavor to find these before considering a radical mastoid operation.

Remarks.—In my experience it has never been necessary to do a radical mastoid operation for an acute otitis media and mastoiditis.

3. I suppose this question refers to chronic suppurative otitis. If a chronic suppurative ear does not show material improvement in two or three months of careful local treatment, I think a radical mastoid operation is indicated.

Dr. Seymour Oppenheimer, New York:

1. This is a very difficult question to answer. If it is an uncomplicated case of acute suppurative otitis media, there is no indication for opening the mastoid. If you mean, however, that it is a case of persistent aural discharge that is rather a different question. The question of the mastoid operation would then depend entirely on the symptoms present, character, and amount of the discharge, temperature, appearance of the fundus of the auditory canal, external manifestations of tenderness, etc., etc.

2. I should not do a radical mastoid operation, but should revise the original mastoid operation, unless the case were one of prolonged suppuration present prior to the original mastoid operation and, even in those instances, the simple mastoidectomy, plus a curettage of the eustachian tube, will ofttimes obviate the necessity of the radical mastoid operation, and in that way lessen the likelihood of diminished hearing.

3. My reply to question No. 2 practically answers your question No. 3. I mean to convey the idea that I should not do a radical mastoid operation unless I were definitely satisfied that the original mastoid cavity had been properly taken care of, and that there existed definite evidence of disease within the tympanic cavity proper. I am, of course, excluding those cases where endocranial symptoms arise, which must of necessity demand immediate interference.

Dr. N. H. Pierce, Chicago:

1. In the ordinary case it requires three weeks for decalcification to occur in the osseous structure of the pneumatic cells. Up to this time (and possibly after) recovery is possible without operation. If an abundant discharge is present at the above named time, the indication for

opening the mastoid is at hand. I rarely open the mastoid before the beginning of the third week in uncomplicated cases.

2. I have very rarely had to perform a radical operation after a simple operation that I have performed myself. If the contents of the cavum are removed, or there is necrosis of the epitympanic space, or because of destruction of the walls of the antrum, or very much depressed external wound and frequent re-infections, I should proceed with the radical operation at any time after the first three months. Of course, there are cases with cerebral complications and involvement of the labyrinth when the radical might have to be done at once.

3. I should open the wound and see if there was not some reason for the continued suppuration in the mastoid before resorting to the radical operation. I have always thought that the radical mastoid operation was a destructive procedure, and should not be resorted to until all other methods have been thoroughly tried out.

Dr. Otto J. Stein, Chicago:

1. If it is an "uncomplicated case," most likely it could be cured without opening the mastoid antrum from without. Almost all acute suppurative cases that require a mastoid operation have some definite symptoms, well known to all of us, necessitating operation.

2. If the case showed no signs of improvement after opening by the simple operation, no doubt evidences of bone caries or necrosis would be demonstrated, and then a more extensive operation would be called for as soon as the condition was shown to be present and not yielding to ordinary measures of treatment. This is always excepting that the symptoms present and persisting are not due to causes transmitted through the tube from the nose, like adenoid or accessory sinus diseases and the like, and also provided no blood dysorasia is present that should be corrected by proper medication.

3. This is answered in Nos. 1 and 2.

Dr. E. A. Crockett, Boston:

1. I practically never operate on a case of an acute otitis media, unless in the presence of symptoms indicating trouble in the mastoid. In the absence of such symptoms, I should allow the ear to run eight to ten weeks. I frequently do so, and find the ultimate result perfectly good.

2. I assume that you are speaking here of an acute suppurative ear, in which case I do not think I should ever do a radical operation. I should simply, in case of necessity, do a second simple operation.

3. A long time, six months at least, and I should perform an ossiculectomy before doing a radical, in any case.

Dr. T. E. Carmody, Denver:

1. Six weeks or two months.

2. Two or three weeks before doing a Weston mastoid or Heath, which might make it unnecessary to do a radical. Should wait after this probably a month or two before doing a radical.

3. Would depend on condition of nose and throat, but probably only long enough to see how it would respond to treatment. Probably two or three weeks.

Dr. J. M. Ray, Louisville, Ky:

1. At least six weeks, depending on the character of the infection. If a capsulatus mucosus infection, somewhat earlier.

2. Several months.

3. I should not perform a radical operation on an acute uncomplicated suppurative middle ear. If there is not good drainage after six weeks, I should advise antrum drainage.

Remarks.—I am of the opinion that an acute suppurative middle ear going from four to six weeks is never an uncomplicated case.

From the answers you will notice that when a time limit is stated, the time varies from two to six weeks.

From the answers to the second question, you will notice that a great number have never found it necessary to do a radical when the simple opening of the antrum had been performed. This is the point that I wish to make: *Early opening of the mastoid antrum prevents the cases from becoming chronic; so the simple operation prevents the radical.*

The answers to the third question vary greatly. It may be that my question was not clear. The patient comes to you and, as a general rule, he has been in the hands of the family physician, who has syringed and swabbed the ears perhaps for weeks and months. The patient gives a history that the discharge has not decreased in the least. How long would you treat this case conservatively before advising the radical mastoid? When I speak of conservative treatment, I wish to include operations on the nose, throat, and eustachian tube.

As to the third question, our practice has been to place the subject before the patient thus: You have a very serious affliction. It may not cause you any serious complications, but there is a chance that the disease will invade some part of the brain and prove fatal. We feel confident in telling the patient that the operation is not a dangerous one, and that in all probability the discharge will cease. On the other hand, as to the effect on the acoustic function of the ear, we are unable to say, as sometimes the hearing is better, sometimes it remains the same, and then it may be a great deal worse after the operation. Our aim has always been to be fair with the patient and not make outlandish statements in reference to his hearing after a radical mastoid.

DISCUSSION

Dr. John W. Murphy, Cincinnati: I think this is one of the problems that is very difficult for us to decide—as to the time we should interfere in chronic or middle ear suppurations. My rule has been, so long as I can see any improvement in the case, *not* to operate. Every case is a law unto itself, and we must study each individual case before we can decide whether it has passed from the acute into the chronic stage. Here the Roentgen-ray is of great importance, as from it we are able to know whether the mastoid has broken down, even before opening or much swelling develops. It is a mistake to wait too long, as while waiting the hearing may become permanently impaired, and it is the object of all

treatment to conserve the hearing of the ear as well as to stop the discharge. No hard or fast rule can be laid down, but I am sure that in many of these cases the treatment could be much shortened by an early drainage of the mastoid cells. In many cases the ear will recover without serious damage, but the time is much longer and the risk too great. I think it is very seldom that these cases, if properly handled, should go on to the point where a radical operation is needed; yet now and then a case goes bad and nothing else seems to give any promise of success. Free drainage must be furnished, and if it is not occurring through ruptured or perforated ear drums, then we must furnish it by draining through the mastoid bone.

Dr. J. E. Brown, Columbus, Ohio: This is an exceedingly interesting subject, and I think it grows in interest when you see the replies to the questions which Dr. Large received from the men to whom he wrote. It shows how differently we interpret questions when we are speaking merely in an academic way. I feel sure that these men would be well informed as to the proper time to operate any case under their own care. The variety in answer shows how many time we do not know whether we have an uncomplicated case. It is the continuance of the inflammation as indicated by the discharge and the other symptoms that begin to tell us when we have complications that we perhaps have not suspected before. Again, the presence of complications even over a considerable length of time does not imply that a complete mastoid drainage must be secured to have good healing. I recall a case last year of a young woman under my care. I was extremely solicitous as to the outcome of the case. I hesitated to do a mastoid. Now, this is a case in which I did no less than three incisions; two of them under anethesia; posterior incision at the margin of the drum membrane, and incisions through Shrapnell's membrane upward and backward along the canal. In spite of that, I had mastoid tenderness, increased temperature, and some indications of infection in the zygoma cells. I made another incision by the canal in front of the short process and up along the anterior superior wall of the canal, and I found that I had been tapping my ear in the right place, for it then cleared up promptly. Thirteen years ago, at the Ohio State Medical meeting at Dayton, I read a paper on mastoid drainage in cases of acute otitis media, in which I made a plea along these lines—namely, that of surgical drainage in cases of acute middle ear suppuration that *appear to be uncomplicated*, but do not go on to recovery in the usual length of time. Our duty should be plain in these cases. I have practically never done a mastoid in a case where the developments of the operation were such as to make me regret having done it. On the other hand, in many cases where I have hesitated to operate I have found the conditions such that I felt I had been negligent in not operating sooner. There is one type of case in which at the inception of the disease, before membrane incision, there is tenderness at the mastoid tip, and perhaps over the antrum, but which disappears after incision. This type of case it is not safe to let pass out of your care until fully recovered, for often a week or two after the activity of infection seems to have passed, the case suddenly develops symptoms of mastoid inflammation. We cannot afford to overlook the aid of the Roentgen-ray picture, in diagnosing or excluding complications in the middle ear cases. I have never had a case in which a radical mastoid was required immediately following an acute middle ear suppuration in an ear previously undiseased.

Dr. J. A. Stucky, Lexington, Ky.: There are two things in favor of early operations. First, to prevent the case from becoming chronic, and, second, to prevent serious complications. Dr. Ingersoll has emphasized

the point I desired to emphasize and that was—to conserve the hearing. A discharging ear that continues for weeks at a time means a thickened and crippled drum membrane, also ankylosis of the chain of bones. An ear that discharges copiously means that something is involved besides the middle ear, probably the mastoid antrum, and the sooner we give the patient posterior drainage the sooner we prevent the complications, such as impaired hearing or meningeal disease. Should there be evidence of otitic meningitis, I should be in favor of doing a radical operation early, in the prepustular stage or the serous stage of meningitis. I should remove the tympanic and antrum roof, so as to give free drainage to the cranial cavity. I do not think we can lay down a hard and fast rule as to when to operate. It all depends on the systemic indications. Everything else being equal, we have two dangers to guide us—the danger of sepsis and the danger of impaired hearing.

Dr. Joseph C. Beck, Chicago: I desire to apologize to Dr. Large for not having answered the questions he put to me in the questionnaire, and for not having any recollection of his having asked me any questions. I want to answer the questions now in the discussion.

I was hoping that Dr. Ingersoll would have said to make use of the roentgenogram as to the indications for operations, for that certainly is a good point. Now, the general symptoms influence me a great deal, as to whether I am going to open a mastoid or not. I mean the general absorptive symptoms, the septic symptoms, and therefore the lapse of no definite time would influence me in deciding when I should do a posterior incision in the mastoid process. The question whether a radical operation should be performed on an acute process, I think, depends on what existed beforehand. If you have a chronic suppuration which had healed up and got an acute exacerbation, then in such cases as that I think it is advisable, unless the general symptoms are very marked, that we do a radical operation at that time. Another thing is the question of hearing.

If the hearing is improving, corresponding to the diminution of the discharge, the case is progressing well. In spite of cleaning out the mastoid process, if the hearing remains bad, there is a pathologic change in the middle ear that is quite likely to indicate that this case is not going to get well by this simple operation. Another thing, the after treatment would influence, or indicate whether the case should be operated on radically following the simple operation, for it must be acknowledged that meddling with the wound and irrigation wil often continue a suppurative process, so that a radical operation has to be done. By closing the primary incision with the separate drainage posteriorly will prevent many cases from becoming chronic through secondary infection.

Dr. Horace Newhart, Minneapolis: I wish to call your attention to one class of cases which, perhaps, has been overlooked, in which the simple mastoid operation is early indicated, and that is those cases of mastoiditis arising in the course of scarlet fever or measles. Here, I think, there is an indication for very early intervention. This is borne out by the fact that a large number of such cases come to us later for a radical operation. When an operation is performed promptly during the course of the acute disease they seldom require secondary intervention. I recall three cases in our clinic at the present time who refused to be operated on during the acute attack of the disease, but who submitted later to an operation. All still have discharging ears, and are awaiting the necessary radical mastoid operation.

Dr. W. T. Michie, Memphis, Tenn.: In none of my cases of acute otitis media have I had to open the mastoid during the sixteen years I

have been in Memphis in private practice, and cured them all. I have had no case of acute otitis media become chronic in private practice. The diet is a very important thing in pus anywhere. The essayist did not say anything about the real problem. I think a great deal of it should be brought home to us. Suppose we had some disease of the mastoid. How long should we wait? We should wait until we thought we are going to die, that is how long we should wait. Here we are still pleading symptoms. Why don't we get at the cause of these things? What is the cause of a mastoid? What is the cause of an abscess of the ear? That is the question. What do you do to keep the pus from forming? It is always cut, cut, cut, and it is the almighty dollar you are thinking about, and God knows it. Now, let me tell you, I used to cut, too, before I didn't know any better. I am young in experience and all of that, but I want to tell you this, that since I found out what I know now I have not operated on a single solitary case of mastoiditis. I know how to feed that individual, which is important. And as long as we are slaves to our stomach we are not going to know. How many in this world eat to live. Darn few of them, and I would put it more definitely if it were not for the ladies present.

A Member: You don't look like you eat to live.

Michie: I never had an ache or pain anywhere and have not been sick in bed in thirty-two years.

A Member: When did you cut out liquor?

Michie: That was nineteen years ago. The sooner you cut out meat, prevent fermentation and decomposition of food, the sooner you will stop operating on mastoids.

Dr. G. C. Kneedler, Pittsburgh: We probably have as many opinions concerning the time to operate for mastoiditis as there are individuals present; however, I cannot approve of what the last speaker has said. Having once decided that an operation was the best thing to do for your patient, and the task being completed, how many have regretted their acts, and, per contra, how many times have we been humiliated by the turn taken in the course of cases that might have been restored to health by operating?

We must admit that pus is present in all cases of suppurative otitis media, and that the mastoid cells are to a greater or less degree involved. To open these cells, under modern asepsis, is devoid of danger. Drainage must be secured for this pus, either through the eustachian tube, drum membrane, or mastoid cells, to prevent its going somewhere else, where greater damage may be done to our patients. Why delay, when we have failed to secure improvement by myringotomy or opening the eustachian canal?

If the temperature returns to normal, but does not remain there (drainage not being perfect), pain does not subside, tenderness over mastoid (but not always present), and with a possible increase in leukocyte findings, do not our guide posts tell us danger is lurking near?

If the case is so near the border line as to make it difficult for a decision, err on the safe side by operating. I should rather operate twice, seemingly prematurely, than delay once too long.

Dr. George M. Case, Elmira, N. Y.: I think the surgeon has to study each case by itself. Also I believe we ought to bring to our use every possible means of diagnosis. Certainly it is wise in every case of suppuration of the middle ear, either acute or chronic, to have a culture taken. If we find staphylococcus infection, operation may be delayed; but if we find pneumococcus or streptococcus infection, I believe we should operate

early. However, there are exceptions to this rule. Recently I had a case of this kind in which the infection was apparently mild. Culture showed staphylococcus, and the operation was delayed until brain complication occurred and the patient died.

The patient was a daughter of a physician, and, although I advised an early operation, he said he "understood that when the infection was of so mild a nature patients usually recovered without an operation," and he refused to give his consent until it was too late. Early operation would have saved this patient's life. There is no fixed rule when to operate and when not to operate. I never did operate when I was sorry; but I have many times condemned myself for the poor judgment I used in not operating on patients earlier.

Dr. Large (closing discussion): I want to thank the gentlemen very much for the interesting discussion. Dr. Murphy, I think, did not understand my question. The question presupposed that the case showed no sign of improvement. By that I mean the amount of the discharge did not decrease in quantity. Another point Dr. Murphy brought up is the question of a radical following a simple opening of the antrum. As a rule, you do not have to do the radical operation, because you have prevented the case becoming chronic. Dr. Stucky brought out a very good point, namely, conserving the hearing. Naturally, if you stop the discharge you will save the hearing. Dr. Brown spoke about mastoiditis. Now, I am not bringing up the point of mastoiditis, because if you have the classic symptoms of mastoids you will have to operate earlier. I mean the uncomplicated cases running from day to day showing no improvement. Those are the cases we should operate on within a time limit. Dr. Ingersoll also brought out the point of conserving the hearing and also the arrest of brain complication. Another point Dr. Stucky brought out, if the discharge is copious, it is not all coming from the middle ear, and you are pretty sure your antrum is involved. Dr. Beck brought out the same point. He said if the ear showed no decrease in the discharge then he would do a mastoid. Dr. Newhart brought out some very important points in reference to hearing and preventing complications.

Dr. Michie, I think, belongs to the Gastro-Enterologic Section. I agree with him that doctors are mighty poor patients. Generally they leave undone those things they should have done and do those things they should not have done. Dr. Kneedler certainly reiterated what I tried to bring out in my paper. As he said, what harm do you do in opening the antrum? The shock to the patient is not as great as it is in a tonsillectomy.

THE VALUE OF BONE AND CARTILAGE TRANSPLANTS IN RHINOLOGICAL SURGERY

WILLIAM WESLEY CARTER, A.M., M.D., F.A.C.S.
NEW YORK CITY

In bringing this subject before you, my chief object is to show by the radiographic history of certain cases that I have operated on, the true value and permanent qualities of bone and cartilage as a transplant in the correction of nasal deformities and defects due to the destruction by disease and traumatism of the bony and cartilaginous framework of the nose. My observations are drawn from a close clinical and roentgenographic study of some of my oldest cases. I began this work nearly seven years ago, and having transplanted bone both with and without periosteum, with and without contact with the living skeletal bones, I feel that a sufficient time has elapsed to say that these cases show accurately the history of autogenous bone and cartilage transplants in the human body. I may state further that this procedure having been subjected to the crucial and ultimate test of application to a number of human subjects, and the results having proved satisfactory, I feel that I may confidently advocate its use both from a scientific and practical point of view.

Animal experimentation is of great assistance in the development of ideas, and it enables one to reach conclusions, approximately correct, as to the value of surgical procedures when practiced on the human subject. The results obtained by the vivisectionist, however, must be verified by the supreme test of application to the human body. In the case of bone transplantation, no statistics as to the value of the periosteum, the vitality of the transplant, its absorption or non-absorption, et cet., obtained by animal experimentation, could be applied to this operation on the human subject, for the reason that in the case of the lower animals it is practically impossible to secure and maintain asepsis, or to keep them quiet until the healing process is complete. Infection, even if it does not cause the transplant to slough out, exerts a marked influence on the vitality of the graft. A certain amount of motion after injury increases osteogenesis in the live, skeletal bones; but upon the transplant itself it has the opposite effect, since it has been suddenly cut off from its normal blood

supply. It must be remembered that the primary, newly developed capillaries which supply the transplant with blood in its new position are extremely delicate, their walls consisting of only one layer of cells, and that they may be destroyed by the most trivial traumatism. This would at least cause a delay in the healing in of the transplant, if not its death and subsequent expulsion. It will be seen, therefore, that while conclusions may be drawn which apply to the animal itself, given the conditions under which animal experimentation must be done, these results can scarcely be considered as furnishing more than presumptive evidence as to what will happen when bone is transplanted in man; or at least statistics as to the fate of the transplant, the value of the periosteum, et cetera, in animals, would be inaccurate when applied to man, in which case conditions are different and rest and asepsis can be easily obtained. Another element of the greatest importance is time. Bone is a tissue comparatively slow in its metabolic processes; therefore, a considerable space of time must elapse before we can estimate with any degree of accuracy the final results and the ultimate disposition of the transplant in the tissues. In case the animals are killed a few days after the operation, no reliable data can be obtained and statistics based on such experience throw no light on the science of bone transplantation. Almost any foreign substance introduced into the body under aseptic conditions can be made to heal in, and it may remain for a long time, but sooner or later it will be expelled, especially if it has resistance to overcome. For this reason no foreign substance can be used successfully to elevate a sunken nasal bridge, for here it is subjected to constant pressure due to the elasticity of the skin. In freshly removed bone and cartilage, on the other hand, we have a live connective tissue capable of being transplanted into another part of the body without losing its vitality, and here it may be used to replace bone and cartilage that has been destroyed by accident or disease.

The interesting question arises as to what part the transplant plays in the process of repair, and what may be the influence of the periosteum, and what the relations are between the transplant and the surrounding tissues. To determine this, a considerable time must elapse after the operation. The cases of my series which I have carefully studied with a view to determining these matters are of long standing and show in the serial roentgenograms very clearly what has taken place. It is my belief that the contradictory conclusions reached by different writers on this

subject are due to differences—in the methods of operating, in the anatomy of the parts operated on, in the site of the operation (allowing mobility or fixation during healing), in the presence or absence of infection and in the time that has elapsed since the operation. Reliable data can be obtained only by studying the progress of a number of cases after the ideal operation. By ideal operation, I mean an operation in which an autogenous transplant has been introduced aseptically in such a position in the human body that perfect immobility can be secured. No part of the

Fig. 1.—Bone with periosteum, transplanted 3 years after operation (congenital deformity).

body is better adapted for securing these conditions than the nose. In addition the abundant bloodsupply to the organ insures to the transplant a maximum of nutrition and resistance to infection.

My work in this field has been confined to transplantations into the nose, with the exception of one case, in which the bone was first grafted into the arm, and then the flap containing the bone was later transferred to the face. In this case the patient had no nose.

Sixty-one cases are included in my series. A number of these have been kept under close observation and roentgenograms have been made from time to time, and at intervals they have been brought before the Larynogologic Section of the New York Academy of Medicine for observation and comment. I do not consider a case of less than a year's standing old enough to furnish data of any value in determining final results.

It is not my purpose on this occasion to dwell on the cosmetic results secured in my work. I will say, however, that no case has been rejected on account of its severity, and that some of my cases

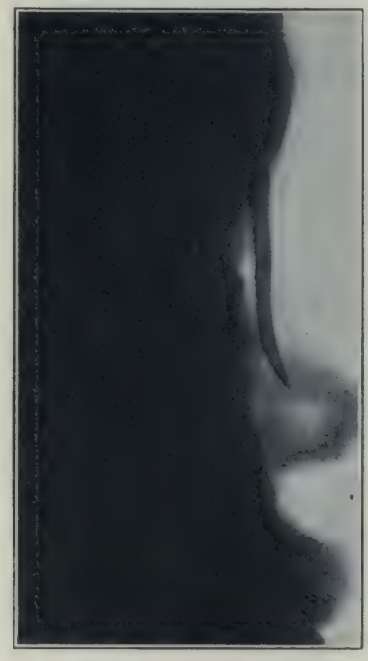


Fig. 2.—Same case 4 years after operation.

have been most difficult. In degree of deformity they have ranged from slight depressions, easily corrected by the transplantation of a spur or portion of a deflected septum removed at the time of the operation, to absence of the entire nose, which was corrected by first transplanting the bone into the arm. In many instances, when the deformity was very great and there was no bony framework left in the nose, my only expectation at the first operation was to raise the bridge of the nose to a moderate degree, my chief object being to establish a bony foundation securely anchored to the frontal bone on which to build up a nose by the

transplantation of more bone and cartilage at some later date. In one severe case I constructed a V-shaped pier from two strips of bone and on this was placed the long dorsal strip. The result in this case was very satisfactory. In others, several strips of bone were superimposed. In these cases, only the outer strip was covered with periosteum. These cases show distinctly the influence of the periosteum on the growth of the transplant.

In estimating the value of my methods from a cosmetic point of view, one must therefore take into consideration the fact that

Fig. 3.—Superimposed fragments of bone in a case of congenital absence of the nasal bones.

a majority of my cases were extreme deformities. I will say that the results have been excellent in many of these, and in others the improvement is marked. In none has the deformity been increased. I can state positively that I know of no other method for correcting this class of deformities so effective as that of bone and cartilage transplantation.

The following were etiologic factors in my series of cases: congenital defects and malformations, traumatism, syphilis, tuberculosis, atrophic rhinitis, the submucous operation and abscess of the septum.

Logically and practically the use of the patient's own tissue for transplantation, the autoplastic operation, is the best method. Here, as I stated in my first communication on this subject, there is no incompatibility between the elements of the transplant and those of the receiving tissues, for the arrangement of the atoms in the molecule of the two is the same. We have in the wound serologic conditions most favorable to immediate healing, which is especially desirable in the transplantation of bone. I have used bone from another patient on only two occasions, and both times the transplant had to be removed; in one seven days, and in the other ten days, after the operation. I will say, however, that

Fig. 4.—Dorsal strip (A) supported by a vertical strip (B). Congenital deformity. (Figs. 5 and 6 show this patient before and after operation.)

in both instances the wound was infected, so that the failure of the graft to take could not be attributed to serologic differences between the transplant and the receiving tissues.

The use of tissue from an animal of a different species, a heteroplastic operation, I have never tried, as I believe that only the best known methods should be applied to the human subject. Many such operations have been reported, but the success of these does not compare with that of autoplastic transplants. Heteroplastic transplants are always absorbed.

I will admit that my early work in this field was largely experimental, but the cases selected were so badly deformed that

I knew no injury would be done even if the transplant had to be removed. The hypothesis on which I began this work was, that bone is a form of living connective tissue, and that a portion detached from one part of the body and transplanted aseptically into another part of the same body would retain its vitality, being first nourished by the serous pabulum which surrounds it, and later by a network of new capillaries developed from the surrounding tissues. This view was contrary to the belief held at that time by histologists, anatomists and pathologists, all of whom believed that a bone transplant was dead tissue; that it was always absorbed, and that new bone was deposited in its stead. My series of carefully observed cases, some of several years' standing, shows that the original transplant is not only not absorbed,

Fig. 5.—Congenital deformity, before operation.

but that it lives, takes part in the local processes of repair, and grows, its growth being subject to the law of functional adaptation. It proves, contrary to the belief of some pathologists, that mature bone is not a dead tissue, but like other tissues undergoes metabolism throughout the life of the individual.

Murphy's view that transplanted bone is merely osteoconductive is altogether at variance with the results shown in these cases.

Nasal deformities naturally fall into two groups: (1) Those cases in which the deformity is due to a dislocation of the anatomic structures of the nose. These may be corrected by the local replacement of the parts and by the use of various mechanical devices. (2) Those cases in which either from accident or disease there has been an actual destruction of the bony and cartilaginous framework of the nose. These cases may be further

complicated by more or less mutilation or destruction of the soft parts and by cicatricial contraction.

Cases belonging to this second group come under our present consideration and may be corrected by the transplantation of bone and cartilage.

I have found the rib will adapted for this purpose. It is well supplied with nutrient foramina; it is properly shaped; is easily accessible, and the removal of a section causes the patient little or no inconvenience. The defect is quickly filled in by bone developed from the stumps of the rib. The end of the rib, with a portion of its costal cartilage, is the part used. This enables me to repair the bony portion of the bridge with bone and the cartilaginous with cartilage. Thus is established a normal condition,

Fig. 6.—Same as Fig. 5 after operation.

as nearly as possible, and the flexibility of the nasal tip is preserved.

Operation by the External Route.—The nasal cavities are cleansed with Dobell's solution. The eyebrows are covered with collodion, after thoroughly scrubbing the entire face with green soap and water, followed by alcohol. The nose and adjacent portions of the face are then painted with tincture of iodin. The right side of the chest is prepared in the same manner. No antiseptic is used after the first incision. I use only freshly sterilized physiologic salt solutiion.

A curvilinear incision, convexity downward, is made between the inner extremities of the eyebrows. This incision extends down to the periosteum. With a spatula-shaped knife, slightly

curved, and having three cutting edges, I then elevate the tissues over the dorsum of the nose down to the tip, and, if the deformity is very great, over the sides as well. If the nasal bones are present, I keep as close to them as possible, endeavoring to strip up the periosteum. After elevating the tissues over the nose, I lift up the semilunar flap made by the first incision and make a horizontal incision through the periosteum and into the frontal bone. Care is taken to cut as deeply as possible into the bone, in order to excite the osteogenesis necessary to bind the end of the transplant to this bone. The so-called periosteum over the frontal bone is not an osteogenetic membrane, and it serves our purpose in this instance merely to hold the end of the transplant in position.

I next proceed to resect about two inches of the ninth rib. This segment is taken from the end of the rib, and consists of

Fig. 7.—Traumatic deformity.

two-thirds rib and one-third costal cartilage. The periosteum on the anterior surface of the transplant is preserved. The segment removed is then split in its transverse diameter, and the outer half, which is covered by periosteum, is shaped to suit the deformity. It is then placed in the bed prepared for it in the nose, the cartilaginous end reaching nearly to the tip, and the upper end being securely tucked under the periosteum, in close contact with the frontal bone. The semilunar flap is then brought down into position and the wound closed with horse-hair sutures.

This external operation is probably better suited to the very severe cases, especially where there is much scar-tissue, than the intranasal method, which I shall presently describe. No effort is made to expel the blood-clot that fills the wound made in ele-

vating the tissues over the nose, for this temporarily nourishes the transplant and later favors osteogenesis.

No manipulation of the part should be permitted, and the case should not be exposed to the Roentgen-ray until the transplant has become fixed in its new position and we are quite sure that its vascular connections have been established. On three occasions, an early Roentgen-ray exposure, (the ninth and tenth day after the operation), seemed to interfere with the healing process. Within forty-eight hours after the exposure to the ray, there was a slight noninflammatory bulging at the site of the incision. An opening was made and there was a discharge of serum. There was no pus formation and the cases progressed satisfactorily afterward. I may be mistaken in attributing the disturbance to the Roentgen-ray, but the clinical connection

Fig. 8.—Same as Fig. 7.

seemed to be so evident that I have been careful since that time not to expose the patient to the rays too soon after the operation.

Recently I have ceased using the external route for the introduction of the transplant except in the very marked and difficult cases.

Operation by the Internal Route.—In this operation the transplant is prepared as I have just described. The left nasal cavity is sterilized and the tip of the nose is raised with the left thumb. The spatula-shaped knife is introduced through the roof of the nasal cavity at a point corresponding to the interval between the upper and lower lateral cartilages. Through this opening, the tissues over the dorsum of the nose are elevated and a pocket is made down toward the tip. The transplant is introduced; then, by pulling on the tip of the nose, the lower end is slipped into its

pocket near the tip. No suture is used to close the wound as the edges naturally fall together. The nostril is packed with petrolatum gauze for twenty-four hours.

This method in suitable cases is far preferable to the external route, as the wound is at the lowest point of the subcutaneous sac and thus drainage is facilitated. There is no external scar, and in my opinion there is less danger of infection, for, it must be remembered that the normal nasal secretions inhibit if they do not actually destroy bacterial growths.

In the cases in which I have used the intranasal route, the results have been excellent, the swelling has been less than by the external method and there have been no cases of infection. If the deformity is marked, several fragments of bone may be superimposed. This is preferable to using one large fragment, as

Figs. 9 and 10 same patient after operation.

small transplants possess relatively greater osteogenetic power than large ones.

In cases of moderate deformity, when there is a septal spur or deflection, this may be removed by the submucous operation and transplanted into the bridge of the nose by the intranasal route. I have, on several occasions, used septal cartilage removed from another patient, in closing a perforation of the septum. In these cases the cartilage seems to serve as a bridge for the mucous membrane.

Operation for Collapsed Alae Due to Loss of Muscular Tone.—The following procedure which I have used only once proved so effective in relieving a patient of this very annoying condition, that I feel justified in describing it: With a very sharp scalpel both alae were split their entire length to the depth of

about three-eighths of an inch. Two thin curved strips of cartilage were then introduced and the wound closed with horsehair sutures. The nostrils were kept distended with petrolatum gauze for several days. When healing was complete, the openings were considerably larger and the patient was considerably relieved.

Time will not permit me to describe all the interesting cases that have come under my observation, and the cosmetic results secured by the transplantation of bone and cartilage. The chief object of this presentation is to call your attention to the value of bone and cartilage as a constructive material, and to demonstrate by serial roentgenographic plates that the vitality of this tissue, when properly transplanted, is not impaired, but that in its new position it lives and grows, its growth being limited by the functional demands of the part.

The following conclusions are based on my interpretation of the clinical and roentgenoscopic evidence presented by cases that were operated on from three to five years ago:

(1) Bone and cartilage present about the same vitality and resistance to infection.

(2) Infection occurring in the wound does not necessarily mean the destruction of the entire transplant, if the infection is quickly controlled. But a part at least of the transplant will be absorbed.

(3) A transplant, stripped of its periosteum, and placed in the soft tissues of the nose, one end being contacted with live bone covered with periosteum, is osteoconductive and osteogenetic and is not absorbed. It does not, however, exhibit the vigorous growth noted in transplants covered with periosteum.

(4) Transplants that are covered with periosteum and which are contracted with live bone covered with periosteum, continue to live and perform their function uninfluenced by their change of position in the body. This is the ideal transplant for use in all departments of surgery.

(5) The periosteum per se does not reproduce bone.

I must conclude therefore, that, while the periosteum is necessary to the vigorous growth of the bone, bare compact bone takes an active part in the production of bone.

In closing, I may add that in bone and cartilage transplantation, I believe we have an aid that will prove of great value in solving some of the difficult problems which confront us in rhinological surgery.

DISCUSSION

Dr. J. C. Beck, Chicago: Every man that practices otology and rhinology ought to be able to do this work. And it is a great credit to Dr. Carter to come here, not alone to show the pictures, but also to show you how it is done. It has been my purpose to do that way with plastic surgery, because I know it is difficult to handle, and yet it is not like removing foreign bodies from the bronchia. It is not dangerous. This work can be done by any one who has surgical skill, provided he acquaints himself with the facts. Heretofore, rhinologists have not been free to show how this is done. Dr. Carter has shown his methods by the use of pictures and I am sure he does these operations at home. So far as the intranasal operation is concerned, that is the operation of choice—the implantation of tissues. I would like to call attention to a method of raising the nose, not with bone, but with bone-forming tissue. That is, the fascia lata. It attaches itself to bone wherever it is placed and becomes hard as bone. That is the method I am now employing for raising the bridge of the nose.

Dr. Wiel, New Orleans: I think this Society is to be congratulated on having this subject presented by the essayist. I think it is a great opportunity to see the roentgenograms. They have been extremely valuable, because it has been a question just exactly how much these bones are nourished. I have personally had very little experience with this nasal plastic work, but I have always been interested in the implantation of foreign parts to replace defective parts, such as septums, and the supposition has been from previous trials that the implantation of any foreign body, such as silver plate, acts as an irritant, and is not a permanent thing, such as bone especially removed from the same individual. Dr. Carter has shown that these bones actually begin to live again through the blood supply they receive from the nose, and that is the interesting point, that this bone thus implanted becomes a living part of the nose, and is not a foreign body placed there merely to make up the deficiency.

Dr. W. W. Carter, New York (closing discussion): I have very little to say in closing this discussion, except that I would like to take issue to a certain extent with the statement made by Dr. Beck that any man who has had a certain amount of clinical training can do this work. He must also have a certain amount of artistic talent to get good results. I think he must have artistic ideals. The correction of nasal deformities does not consist merely in the placing of tissue beneath the skin. Each case is different from the others, and a certain amount of subcutaneous work must always be done in each case, which is not always mentioned in reviewing the case and which does not show in the roentgenogram. I think a man should go at this work very carefully before he attempts any very difficult case. If he does not he will bring not only discredit on himself and on the operation, but also on the specialty which he represents in the locality where he lives. Now, I would just like to refer to a case that happened just six weeks ago. A physician wrote me a letter stating that he had a certain case and that he wanted some information in regard to it, as he wished to operate on the patient himself. I sat down and very conscientiously wrote him a complete description as to how to do the operation and referred him to my various writings on the subject. Just before I left New York to come to this meeting, I received a letter from him stating he had operated on the patient, that the operation was a failure, and that he had subsequently offered to bring him to New York to me, but that the patient had disregarded his advice in that respect, and had left for the East without consulting him, and I

don't blame him. Now, it is unnecessary for me to suggest that this patient did not come to me. If the physician had received from Dr. Carter explicit directions as to how to do the operation and he had attempted it and failed, Dr. Carter and his methods would be the very last things this patient would want to see or hear anything about. And I think in a case of that kind it brings discredit on the method and on me. This field of rhinologic surgery consitutes a specialty in itself, and I firmly believe that this work should not be attempted by those who are not qualified either by natural endowment or by clinical experience. I am not making these remarks from a selfish point of view, but because I am interested in this kind of work, and I want to see it well done. I thank you for the interest you have shown in my presentation of this subject.

69 West Fiftieth St.

PRIMARY CARCINOMA OF THE MIDDLE EAR, WITH REPORT OF CASE

HORACE NEWHART, A.B., M.D.
MINNEAPOLIS

Carcinoma of the middle ear, because of its very rare occurrence and extremely bad prognosis, has not been a favorite topic in medical literature. In view of an ever-increasing interest in malignant neoplasms, and recognizing the successes in other departments of surgery through early diagnosis and aggressive treatment of cancer, a brief presentation of this infrequently described condition, inspired by the observation of a recent case, is of interest at this time.

Primary carcinoma of the middle ear was long regarded as one of the curiosities of medicine. As early as 1886, Kretschmann[1] collected sixteen cases. In 1899, Zeroni[2] reported five cases of carcinoma of the ear, three of which, however, were of the external ear or of the canal. His very exhaustive study of the literature yielded 121 cases of carcinoma of the ear reported between the years 1804 and 1899. Of these, a moderate number were primary middle ear carcinomata.

During twenty-four years Bezold[3] saw but four cases of carcinoma, three of the external ear and one primary in the middle ear, out of 20,000 otologic patients.

Levesque,[4] reporting a case in 1910, says that in all the preceding literature he could find less than fifty authenticated cases. He cites Dupau, who estimates that there occurs but one case of malignant disease of the midlle ear to every 10,000 cases of chronic otorrhea. Milligan,[5] discussing a report of a case by Whitehead, said that in the records of the London Hospital averaging 200,000 patients per annum for a ten year period, not a single case of carcinoma of the middle ear could be found. For the same period more than thirty cases of malignant disease of the external ear were recorded.

---

1. Arch. f. Ohrenh., 1886, **24**, 231. Ueber Carcinoma des Schläfenbeines.
2. Arch. f. Ohrenh., 1899, **48**, 141-190. Ueber das Carcinom des Gehörorgans.
3. Cited by Treitel, Ztschr. f. Ohrenh., 1898, **33**, 152.
4. Gaz. méd. de Nantes, 1910, **28**, 89-93. Des Tumeurs malignes primitives de l'oreille moyenne.
5. Proceedings Royal Soc. Med., London, 1907-8, Otol. Sec., discussing report of Whitehead's case.

In the literature accessible we have been able to find, since the date of Zeroni's monograph (1899), reports of thirty-four cases of middle ear carcinomata. This includes, however, several mentioned in discussions, but not reported *in extenso*.

American otological literature on this subject is extremely scanty. All told we have found but eight[6] cases published in the United States.

In order to determine the relative frequency of this disease in America, and the approximate ratio of reported cases to those not reported, we recently sent inquiries to a number of otologists throughout the country, asking how many proven cases each had seen in his practice, or in consultation, and how many he had published. Two hundred and forty-seven replies have already been received. One hundred and ninety-two otologists, many of them men of very extensive practice, replied that they had never had a case. Forty-five men returned a total of fifty-one cases, two having had five cases each.* This fact suggests the possibility of a very uneven geographical distribution of the disease. Only two of the forty-five cases had been published.

It is probable that this number represents only a small proportion of all cases which have occurred during the period covered by these observations, many never having come to the attention of a specialist, and many doubtless having died without a correct diagnosis.

While occurring most frequently after the age of 40, Alexander[7] saw a case in a boy of 17, Leidler[8] reported one at 17, Nager[9] one at 19, and Knapp[10] one at 20.

Both sexes are equally liable to the disease.

Concerning the etiology, the most constant factor which may be regarded as possessing any causal relation is the purulent

---

* Since the reading of this paper there have been received sixteen further replies, one reporting no cases, and fifteen reporting a total of twenty-one cases.

6. a. Whiting, F., N. Y. Eye and Ear Infirmary Reports, 1898, **6**, 91-98. Carcinoma of the Mastoid and Auricle Originating in the Tympanum or Antrum, Consecutive to Chronic Otitis Media.
   b. Gerber, Arch. Otol. N. Y., 1904, p. 482. Three Cases of Tumor of the Ear.
   c. Behrens, Trans. Am. Laryn., Rhinol. and Otol. Soc., 1905. A Case of Epithelioma of the Middle Ear.
   d. Halstead, discussing above.
   e. Lutz, discussing above.
   f. Knapp, discussing above.
   g. Braislin, Ann. Otol., Rhinol. and Laryngol., 1907, p. 90. Epithelioma of the Ear.
   h. Goldbach, Laryngoscope, 1914, p. 128. Squamous Epithelioma of the Middle Ear and Mastoid.
7. Die Ohrenkrankheiten im Kindesalter, Leipzig, 1912.
8. Das Mittelohrkarzinom im Lichte moderner Krebsforschung, Arch. f. Ohrenh. 1908, p. 177.
9. Ueber die Bildung von Labyrinthsequester bei Mittelohrkarcinom, Verh. d. Deutsch. Otol. Gesellsch., 1908, pp. 130-135.
10. Cited by Behrens, Trans. Am. Laryngol., Rhinol. and Otol. Soc., 1905.

discharge of a chronic otitis media. This was present in over 85 per cent. of all cases. Many gave a history of suppuration from infancy or early childhood.

Treitel[11] and Danzinger[12] reported cases which appeared without a previous chronic discharge, but followed closely on an attack of influenza, with only an acute otitis preceding the symptoms of carcinoma.

A history of trauma, from four weeks to nine months before the onset of symptoms was noted in two cases reported by

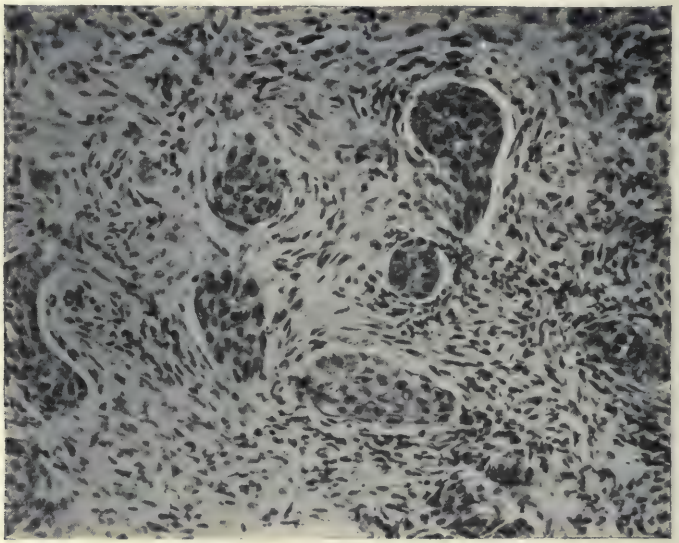


Fig. 1.—Hematoxylin and eosin. Photomicrograph of tissue removed by operation. Nests of epithelial cells embedded in dense connective tissue. No epithelial "pearls" are seen. The cells definitely resemble those appearing in the secretions at post-mortem. × 1700.

Milligan,[13, 14] in one reported by Wilkin,[15] and in one case reported by Dalby.[16]

Heredity plays the same rôle as in carcinoma of other parts of the body.

Of all symptoms, the most constant and usually the earliest, is pain referred to the meatus or to the postauricular region. Next in frequency is the presence of bleeding granulations in

11. Ueber das Karzinom des Ohres, Ztschr. f. Ohrenh., 1898, **33**, 152.
12. Beitrag zur Kenntniss des Felsenbeinkarcinoms, Arch. f. Ohrenh., 1896, **41**, 35.
13. Epithelioma of the Middle Ear (Traumatic), Proc. Roy. Soc. Med., London, 1907-8, I.
14. Carcinoma of the Middle Ear, Proc. Roy. Soc. Med., London, 1911-12, Otol. Sec., p. 26.
15. Report of Two Cases of Epithelioma of the Middle Ear, Treated with Pyoktanin, Jour. Laryngol., Rhinol. and Otol., 1898, p. 343.
16. Cancer of the Ear, Lancet, London, 1892, p. 497.

the middle ear or meatus. Facial paralysis occurs relatively early in most cases. Mastoid tenderness and swelling with a superficial ulceration appear in the more advanced stages.

Impairment of hearing is proportional to the amount of involvement of the cochlea, either by direct extension of the carcinomatous process, or through inflammatory changes. In a few cases (Nager[17] and Broeckaert[18]) some degree of hearing was present until within a few days of death, as in the case herewith reported. Symptoms on the part of the static labyrinth may appear at any time during the progress of the disease, but

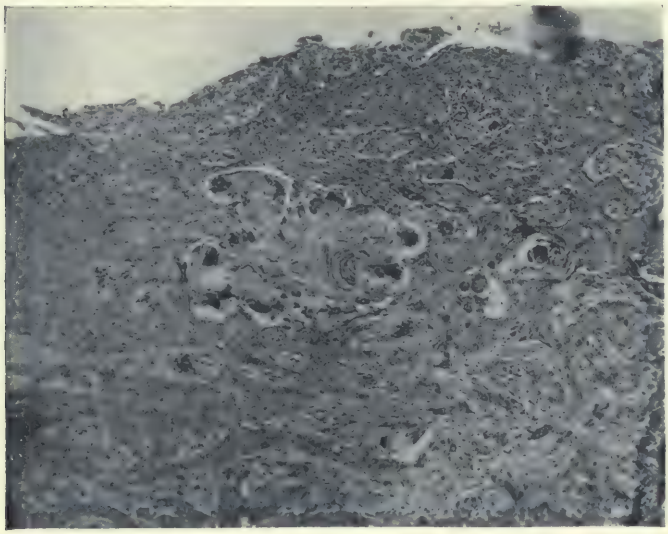


Fig. 2.—Hematoxylin and eosin. Microphotograph of section showing dense hyalinization of the epithelial "pearls" with attempt at inclusion by foreign body giant cells. × 750.

they are frequently very late, for the labyrinthine capsule is especially resistant.

Cachexia is a common characteristic in spite of the fact that there is practically never an involvement of organs which have to do with nutrition. This is explained by the disturbances due to the pain and to the poisons produced by the neoplasm itself.

As to the microscopic pathology, it was found that the cases in which the histologic findings were given were epitheliomas of the squamous-celled variety. There was one notable excep-

17. Ueber die Bildung von Labyrinthsequester bei Mittelohrkarcinom, Verh. d. Deutsch. Otol. Gesellsch., 1908.

18. Carcinome epithelial de l'oreille moyenne, Revue Internat. de Rhinol., Otol., etc., 1898, **2**, 243.

tion in a case described by Lange[19] which proved to be a cylindric-celled epithelioma.

The question as to the method of origin has not been settled. The great majority of middle ear carcinomas can be accounted for by the extension into the tympanic cavity of the epithelium from the external canal. In consequence of the traumatism to this epithelial tissue, by the chemical irritation of a purulent discharge, there is evolved a carcinoma. But in those cases which originate in a closed tympanic cavity we must assume a metaplasia of the cylindric-celled lining membrane into the squamous-

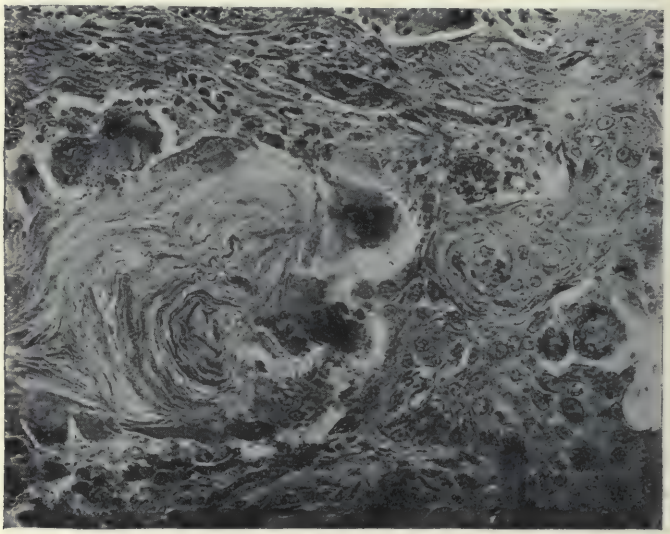


Fig. 3.—Hematoxylin and eosin. High power view of same section. × 1700.

celled variety, or conclude that the neoplasm is derived from glandular elements within the cavity.[20]

Invasion of the bone takes place from the tympanum and its destruction goes on rapidly with the formation of irregular sequestrums. The bone is destroyed partly by pressure and partly by obliteration of its blood supply. The labyrinthine capsule, because of its compactness, is particularly resistant to the new growth. The dura also presents a decided barrier to its extension to the brain. Though covered with the so-called granulations, the dura is frequently found intact on its inner surface.

19. Ein Fall von primärem Zylinderzellenkarcinom des Mittelohres, Ztschr. f. Ohrenh., 1904, **46**, 209.
20. Panse, Pathologische Anatomie des Ohres, Leipzig, 1912.

The adjoining lymph glands are enlarged, but microscopically they give the picture of an inflammatory reaction, with small-celled infiltration. Practically never do they show the presence of carcinoma cells.[21]

With the advance of the new growth, neighboring organs become involved. The parotid gland and mandibular joint are among the first to be invaded. The lateral sinus is not infrequently surrounded by the carcinoma and obliterated, and in advanced cases the meninges, brain, and cranial nerves are attacked.

Metastases to other parts of the body are very unusual. The lungs and mediastinum were found the seat of metastases by Treitel[22] and Lange.[23]

Carcinoma of the middle ear runs a rather rapid course, though its progress is much slower than sarcoma of the same region. In any given case it is impossible to determine the exact duration, for one cannot fix on the time the neoplasm started. However, the interval from the first symptoms of pain to the time of death is from eight to eighteen months, with one year as the average duration. Death is caused by meningitis, brain abscess, hemorrhage, exhaustion, or by the direct extension of the growth to the vital centers.

An early diagnosis can be made only with the microscope. For this reason every aural polyp, especially in persons over 40 years of age, should be microscopically examined. Furthermore, all suspicious granulation tissue removed in the course of the radical operation should be carefully scrutinized because of the possibility of malignancy.

Radiography of the mastoid region offers large future possibilities as an aid to diagnosis by the early recognition of destruction of bone before the growth has extended far beyond the tympanic cavity.

Luetic disease of the bone must be excluded by the history and by serum diagnosis.

The prognosis is hopeless in all cases not operated on. Stein[24] reported a case in which a polyp was removed with the snare. The microscope proved it to be a carcinoma, but there was no recurrence. Jansen[25] operated on a patient who lived

21. Leidler, Das Mittelohrkarcinom im Lichte moderner Krebsforschung, Arch. f. Ohrenh., 1908, p. 177.
22. Treitel, Ueber das Karcinom des Ohres, Ztschr. f. Ohrenh., 1898, **33**, 152.
23. Lange, Cited above.
24. Stein, Beitrag zur Lehre von den Geschwülsten des Ohres, Inaug. Dissert. Königsberg, 1896.
25. Jansen, Verh. der Deutsch. Otol. Gesellsch., 1897, p. 241.

eight months without recurrence, dying of an intercurrent disease. Whitehead[26] reported a case which showed no recurrence twelve months after operation.

From the nonfatal cases in the literature, we must conclude that the prognosis is favorable in proportion to the earliness of the diagnosis and the thoroughness of the operative procedure.

The only effective treatment consists in thorough operation. Some recent writers, including Brindel[27] and Zebrawski,[28] advise against all interference. This view, however, is not

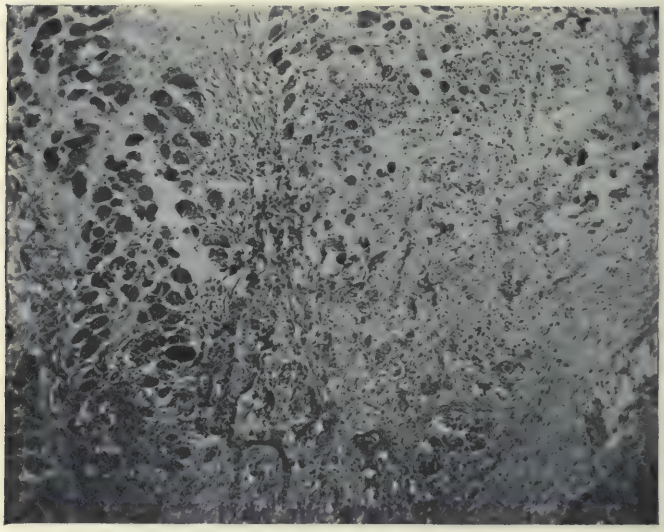


Fig. 4.—Hematoxylin and eosin. Groups of epithelial cells invading striated muscle. × 750.

shared by the majority of otologists. As early as 1898, Heyer[29] successfully resected the temporal bone for a carcinoma primary in the external ear, but with extensive bone involvement.

Dr. Holinger has recently written me in regard to an unpublished case of a patient, aged 38 or 40, on whom he operated ten years ago, and who is still alive. Dr. Carl L. Larsen of St. Paul, in November, 1914, operated on a man aged 50, doing a radical mastoid, followed by the actual cautery, and recently advised me that his patient has had no recurrence. This case has not yet been published.

---

26. Proceed. Roy. Soc. Med., London, 1907-8, Otol. Sec., p. 34.
27. Rev. hebd. de Laryngol., etc., 1912, I, 689-701.
28. Monatschr. f. Ohrenh., 1912, p. 217.
29. Ueber einen Fall von Ohrkarcinom behandelt mit Resection des Felsenbeines, Deutsch. Ztschr. f. Chirurgie, 1898-99, I, 522.

For the sake of relieving suffering, the considerate surgeon will usually decide to operate in all but the most advanced cases. And the fearless operator, who is so fortunate as to see his case sufficiently early, will operate with some reasonable hope of a successful outcome. We must regard as inoperable those cases in which there is extension within the cranium, or into the soft parts of the neck.

Thus far we have been unable to find, either in the literature or through inquiry, any positively successful results from the use of radium or the Roentgen-ray in treating carcinoma of the

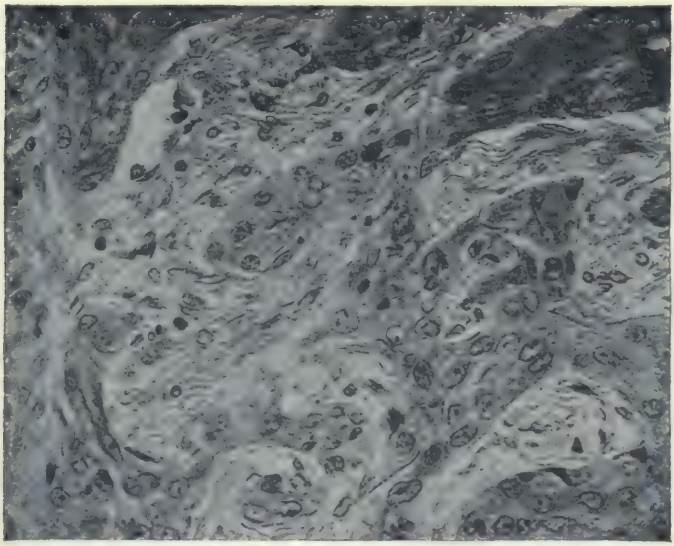


Fig. 5.—Hematoxylin and eosin. High power view of morphology and arrangement of epithelial cells with intervening connective tissue. A mitotic figure in center of picture. × 2000.

middle ear. Urbantschitsch[30] has recently published a report of several cases of ear diseases successfully treated with radium, including an epithelioma and a sarcoma of the external ear.

Deep Roentgen therapy by improved technic, when employed as an after treatment following operation, promises much for a larger percentage of cures of this disease.

Case 1.—Mrs. E. C., aged 65, was first seen May 12, 1915. She complained of severe pain within the right ear and of an unpleasant discharge with occasional bleeding from the canal. Family history was negative. There was no previous ear disease. Present illness began January, 1915, with an attack of influenza,

30. Urbantschitsch V. Ueber einige mit Radium behandelte Ohrenfälle, Arch. f. Ohrenh., **96**, 15.

when she felt a fullness in the ear, followed by pain which was relieved in a few days by the onset of a discharge. She felt variable pains in the right ear until April 15, when she noticed that the discharge was at times more copious and consisted largely of blood. The pains became more severe, but were relieved for a time by each hemorrhage.

Examination showed a woman well past middle age, somewhat cachectic, extremely restless and worn with pain. Weight 145 lbs.

The right canal was partly filled with a dark, fetid discharge. The inner fourth of the posterior superior meatal wall was red and slightly swollen. All but the anterior inferior third of the drum-head was destroyed. The ossicles could not be found.

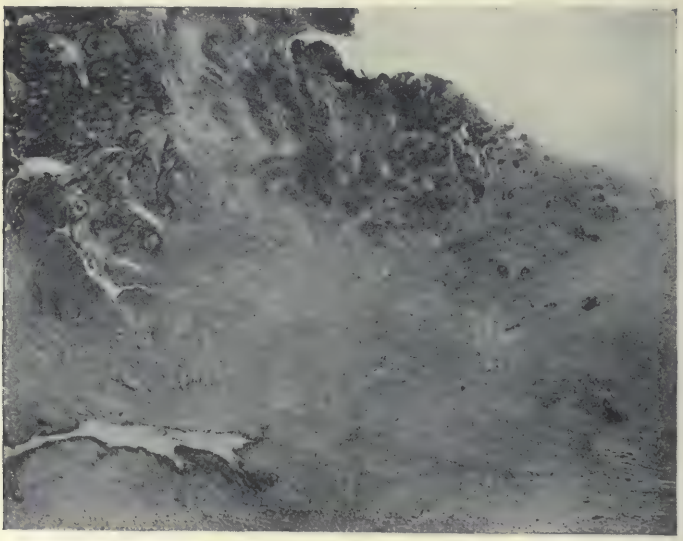


Fig. 6.—Hematoxylin and eosin. Low power view of bony trabeculae in one portion of section and masses of epithelial cells in another portion with intervening inflammatory connective tissue. × 35.

Within the tympanic cavity, but not extending into the canal, were dark red, bleeding granulations. The probe revealed exposed bone in the posterior portion of the tympanic cavity. The mastoid showed no swelling or redness, but was tender over the antrum and tip. There were no enlarged cervical or auricular lymph glands.

The left ear was normal.

Hearing distance for the right ear (left ear excluded with the Barany apparatus) was ten feet for the voice and two feet for the whisper. Weber was lateralized to the right, Rinné was negative on the right, positive on the left. There was no spontaneous nystagmus. The fistula test was negative.

An immediate radical mastoid operation was advised. The patient was referred to Dr. J. G. Cross for a general examination. He reported a blood pressure of 190, with a chronic nephritis, and advised a postponement of the proposed operation.

May 26, we observed a slight facial paresis on the right side involving all three branches. The pains became more intense.

The Wassermann reaction was negative. A roentgenogram made at this time showed extensive destruction of the bone.

Malignancy was suspected and we again urged operation to relieve pain.

May 28, operation, St. Barnabas Hospital. The primary postauricular incision was attended by excessive bleeding. The knife on cutting through the periosteum plunged deeply into the

Fig. 7.—Post-mortem photograph showing extensive destruction of temporal bone.

mastoid, which was filled with highly vascularized granulations interspersed with small irregular sequestra. The large cavity was thoroughly cleansed with the finger and curette. Surrounding the labyrinthine capsule a large sequestrum was found which could not be removed without destruction of the labyrinth. It was therefore left. A small area of dura covered by granulations was exposed over the middle fossa.

A diagnosis of inoperable malignant growth was made. The patient's age and poor physical condition and the involvement of the dura rendered further procedure unwise.

The patient rallied rapidly from the operation and on the second day stated she was entirely free from pain. The facial paresis completely disappeared by the fifth day.

The wound healed rapidly and the patient's general condition improved noticeably, as a result of the rest which was now possible.

July 6. The right ear perceived the whisper at eight inches. Weber was lateralized to the right. The course was uneventful until August 3, except for the occasional return of pain and slight hemorrhages on change of dressing.

August 3. Tumor masses appeared deep in the auricular canal. The superior branch of the facial nerve again showed paresis.

August 6. The facial paresis now extended to all three branches. Because of a return of very severe pains a second operation was decided upon. Tested before the operation, the right ear perceived the conversation voice at fourteen inches (exclusion apparatus in left ear).

Second operation August 7, Dr. E. S. Strout assisting. The original incision was opened. The cavity in the bone had grown and was filled with granulations and sequestra which were removed, except the large movable mass corresponding to the labyrinthine capsule. The facial nerve was found exposed in the cavity for a distance of more than one centimeter. The dura over the middle fossa covered by granulations was exposed over an area with a diameter of nearly 2 cm.

August 8. The patient again rallied promptly from the operation. The pains diminished.

August 10. The C_4 fork and the voice were perceived through the dressings. There was no spontaneous nystagmus and no nausea. Weight was 123 lbs.

September 1. The patient left the hospital for a short walk. The pains increased, but were controlled by codein. The cachexia was very noticeable. The facial paralysis was now complete. Patient perceived the loud voice at eight inches, left ear excluded.

September 6. A very severe spontaneous hemorrhage occurred from the canal and the postauricular wound, but was controlled by a firm packing.

The course so far was afebrile. Temperature on this date was 99.2; pulse 112. The patient being very restless from pain, morphin was necessary for the first time.

September 9. There occurred a very severe hemorrhage on change of dressing. Pulse 156, irregular.

September 12. During the night there was a severe spontaneous hemorrhage. The patient was very weak and delirious.

September 13. Death.

Pathologic Examination by Dr. Harold E. Robertson, Sept. 13, 1915.—External examination of the body showed a rather poorly nourished adult female, 163 cm. in length. Pupils dilated unequal,

the right measuring 7 mm., the left 6 mm. in diameter. Slight general subcutaneous edema. Multiple fine cutaneous hemorrhages in the skin over the left shoulder, and to a less extent over the right shoulder and right side of abdomen. Behind the right ear is an excavated ulcer 6.5 cm. in length, 3 cm. in width, and 2.7 cm. at its greatest depth. Its inner surface is covered by a smooth granular membrane, from which bony points project and from which, on palpation, pockets of thick yellowish pus are discharged. This granular-appearing ulcer extends downward toward the parotid gland and also evidently involves the bony parts immediately adjacent to the external auditory canal. The skin of the right side of the face is smoother than that of the left, and the right angle of the mouth is slightly depressed.

Internal examination of the body shows advanced degree of arteriosclerosis with chronic diffuse nephritis and fibrous changes in the myocardium. Both tonsils contain thick yellowish material embedded in the crypts, but neither tongue, pharynx, or larynx show any evidence of tumor or ulcerative processes.

On removing calvarium, the longitudinal sinus fails to show any evidence of thrombosis. On removing brain from skull, on the inside of the dura at the junction of the squamous and petrous portions of the right temporal bone is an oval raised plaque, measuring about 3 by 2 cm., and corresponding in position to the excavated area described above. This plaque is composed of firm tissue having a slight granular appearance and yellowish tinge, resembling markedly the tissue which lines the entire ulcer. Pieces of dead bone are readily removed from the base of this ulcer, and the process is now seen to involve the facial nerve, the right inferior maxillary bone, the posterior portions of the right parotid gland, and the outer portions of the bony labyrinth on the right side. In all of these locations the bones are embedded in a granulation-tissue-like mass which had evidently partially destroyed the periosteum, creating small sequestra during the process. There is no evidence of any involvement of adjacent lymph nodes, or of the inner surface of the calvarium, or other regions of the neck, or face, or any portion of the brain.

Microscopic examination of portions of the tissue involved in this ulcerated area shows masses of epithelial cells resembling in arrangement and morphology squamous epithelium. Typical pearl formation is illustrated at several points. Many of these pearls

have become definitely hyalinized and are surrounded by nuclei of large giant cells. In other portions these cell nests are extremely active in growth, as indicated by the large proportion of mitotic figures and their invasion of muscle in some areas and in other portions of lobules of the parotid gland.

Examination of the specimen removed at the operation shows cell nests corresponding in every respect, except for the absence of epithelial pearls, with the tissue just described.

The diagnosis in this case is clearly a squamous cell carcinoma. At the post mortem its point of origin could not be definitely determined, but the very marked involvement of the structures of the middle ear strongly suggests this site as the origin of this growth.

DISCUSSION

Dr. Frank Todd, Minneapolis: I will spend a few moments in the discussion. Dr. Newhart has brought to our attention the fact that this disease is much commoner than we, relying for our information on the reported cases, supposed. I have seen two cases, one in consultation and this case that I shall report, which emphasizes Dr. Newhart's point that foreign tissues should be examined in cases in which there is chronic middle ear suppuration, and even when we do not suspect the existence of a malignant growth. The pictures which I shall pass around will illustrate this point. The case was seen July 22, 1900. The patient, Mrs. Young, aged 58, the wife of a farmer, resided in South Dakota. She had had chronic suppuration of the right ear, with discharge for the past four years, and the discharge had been accompanied by great pain and swelling behind the auricle. Facial paralysis began about five months previously. On examination an ulcerated concha was found. The auricle was swollen. The external auditory canal was closed from swelling. The mastoid was tender, but not swollen. Radical operation was performed on July 23, and very much necrosed bone was presented. The mastoid was removed. Incision had to be extended down at the neck, exposing the inferior maxilla, and the petrous portion of the temporal bone came away. Despite this extensive involvement and necrosis, no examination of the material was made. It should have been made, of course, but it was not. This was sixteen or seventeen years ago. The wound healed gradually and the discharge ceased. The patient returned again in October, 1902, with a fistula behind the auricle, which she stated had been present for six months. She was having severe headaches, fever and chills, and was very much emaciated, and on November 1, an exposure was made, tissue taken, and it was found to be carcinomatous. A very large area of necrotic and granulation tissue was present. You will see in the picture the case as it appeared shortly after the last exposure. The patient died July 26, 1903. I noticed that the essayist stated in his paper that these cases extend over a period of eighteen months as a maximum. This case, from the time I first observed it until death took place, covered a period of three years. Of course, we had no examination of tissue at the time of the first operation, but it is evident that it was carcinomatous.

Dr. Joseph Beck, Chicago: The interesting point in Dr. Todd's case is that it lasted so long. I have here a picture of a man on whose ear I operated about ten years ago, secondary to the use of arsenic paste. He lived almost three years from the beginning of the development of the epithelioma. As soon as the parotid becomes involved in these cases they spread very rapidly. I have had no experience with carcinoma of the middle ear. I have had, however, a case of primary endothelioma of the middle ear. That case was diagnosed at first microscopically as carcinoma. It is important that two or three, or more, microscopic examinations be made, and by different laboratory men; for the interpretation of microscopic examinations may be misleading. Several points interested me in this paper. I wondered why there was not a section made of the bone to see what the involvement of the bone was. You will find in a review that I made of twenty years' work of carcinomas, in all parts of the body, that arteries and cartilage are not involved, but the bone is, and it would have been very interesting to see a microscopic examination of the bone. Another point is the cause of the hemorrhage. You should have seen in the postmortem some large arteries, like the carotid or jugular bulb, from which it would have originated. The question of lymphatics not being involved has been brought out by Reik and others, which shows that the lymphatic distribution from the middle ear and external canal is very scanty, and that is an encouraging thing. To make an early diagnosis, one is to suspect in an old person with polyp formation in the canal that it is cancerous until it is disproved particularly by the microscope.

Dr. G. C. Kneedler, Pittsburgh: Mrs. H., aged 28, with a history of chronic suppurative otitis media in left ear since childhood and practically a continuous discharge since, came to my office suffering from pain in left ear and over greater portion of left side of head. There was also some tenderness over mastoid on pressure. Inspection showed granulation tissue almost filling the canal, and with some discharge around the mass. Slight edema over mastoid, but no redness. At the Eye and Ear Hospital, I removed a section of the protruding mass, which proved to be carcinomatous. The patient did not return for about four months, when the condition was quite grave, with pain so severe that she had resorted to the use of morphin for relief. The swelling, postauricular as well as preauricular, had become very extensive and extremely painful to the touch. With my colleagues, whom I consulted, I advised noninterference. The patient passed from our hands to a dispensary, where she was operated on, but died shortly afterward.

I believe, as Dr. Beck has suggested, that two or three examinations of tissue should be made before a positive diagnosis of carcinoma be given. Take the case of a patient of my own, on whom I operated with the cautery about two and a half years ago. The patient is now well and free from his trouble, and though one examination of tissue proved to be cancerous, I now believe the case was not malignant. This might have been verified, had there been two or more sections made at that time.

Dr. Newhart (closing discussion): I appreciate very much the discussion, which is extremely interesting. This case I reported, believing that a great many such cases are overlooked until too late. Dr. Beck's suggestion as to the advisability of detailed pathologic examinations is extremely important and applies in this case. I did not have time to mention in the paper the fact that we did send specimens from the first operation to two different pathologists, who gave negative reports. This perhaps, was not due to faulty technic or ignorance, but possibly they did not

have the right sections. I expected to find an erosion of a larger vessel, but this was not disclosed at autopsy. In regard to duration, I refer only to those cases which originate in the middle ear, and gave the time as that presumably from the involvement of the middle ear and after extension into the bone, when the course is rapid. Epitheliomata ordinarily progress at a rather slow rate. Dr. Kneedler's case is very instructive and throws considerable light on the possibilities of treatment. After all, a thorough treatment with the cautery following operation offers a considerable hope of recovery unless the condition is too far advanced. In this respect my own case, I have felt ever since the postmortem, represents a failure. Had this patient been younger, and had we operated fearlessly when she first come into our hands, I believe she would have stood a good chance of recovery, for the extension was not great.

OSTEOMYELITIS OF THE SKULL

REPORT OF A CASE WITH PANSINUSITIS, TUBERCULOSIS, SYPHILIS, BRAIN ABSCESS, AND BILATERAL MASTOIDITIS WITH ADDITIONAL OPERATED FRONTAL SINUS CASES

H. A. Beaudoux, M.D.
ST. PAUL, MINN.

Cases of osteomyelitis of the skull, originating from frontal sinusitis, seem to be of very rare occurrence. Therefore I feel, even though I may not have anything new to add as regards the etiology, bacteriology, and treatment of these cases, that this particular case, which was under my care from Nov. 6, 1912, to June 9, 1914, presents a series of sufficiently interesting experiences to arouse at least discussion of this unfortunate malady. This is my only excuse for bringing this matter before you.

Osteomyelitis of the temporal bone, as a sequel to otitis media suppurativa, occurs, and has been reported from time to time, in a comparatively large number of cases, though it has often been overlooked by the operator as such. Moller, in the *Jour. Am. Med. Assn.*, February, 1908, reports a case of osteomyelitis of the temporal bone, with fifteen cases found in the literature. C. W. Richardson of Washington, D. C., reported a case of osteomyelitis of the temporal bone in June, 1900, at the A. M. A. meeting, and he reported a similar case Nov. 12, 1903, at the Academy of Medicine.

I am not able to find in the literature at my command any cases of osetomyelitis of the frontal bone originating per se from sinus disease, except the well known cases following surgical operations upon the sinus and particularly those of a nonradical nature. Therefore, I consider this case the more interesting because of its apparent rarity.

Case 1.—The patient, a man aged 49, presented himself for examination Nov. 6, 1912, with a temperature of 101, pulse 98, complaining of excessive bronchial excretion of a purulent nature and of a frontal pain located just in the medial line of the forehead, between the sinuses. He gave a history of having had a diagnosis made twenty-five years ago of a tubercular involvement of the right lung. After coming west in search of better health, he had repeated hemorrhages for several years, but had been in apparent good health for more than twenty years, with the exception of sinus disease, for which he had frequently consulted rhin-

ologists and had received more or less satisfactory treatment. He had been advised during the past fifteen years that a radical operation would be necessary to obtain a cure; but, being of an inquisitive and rather fastidious nature, he had gone into the subject very thoroughly and had refused any operative interference lest his personal appearance be impaired.

Physical examination revealed an involvement of both lungs, some emphysema, a chronic purulent bronchitis with excessive quantities of pus, and temperature running from 100 to 101 daily.

Pansinusitis, with an exception of the right sphenoid was confirmed both by irrigation and the Roentgen ray. A general atrophic rhinitis was present. The roentgenograms showed no evidence accounting for the sharp pain in the lower frontal region, from which he complained severely at times. Repeated Wassermanns were made to ascertain the presence of syphilis, as I had several reasons for suspecting its presence, but were found to be negative. Careful search for bacilli was made then in two separate laboratories and subsequently, without ever being found. Von Pirquet and tuberculin tests were unsatisfactory.

Notwithstanding these negative results, provocative tests for syphilis were made, as well as the luetin test, the latter giving only a faint reaction and the former gave an XX positive.

The *bacteriological examination* showed the infection to be staphylococcic, and staphylococcus albus and micrococcus catarrhalis, together with an unidentified organism, were found on repeated examination.

Although the patient's pain improved under irrigation of the frontal sinuses and antra, he was given five injections of salvarsan, administered by Dr. Paul Cook one week or so apart.

Blood examination, which showed a leukocytosis of 14,000, was stimulated by autogenous vaccine, with only a slight rise of from eighteen to twenty thousand, and because of this, it was decided to discontinue its use.

The right antrum flowed copiously and produced such quantities of pus at each irrigation that on November 12 it was decided to perform the radical operation through the canine fossa, as an intranasal operation had been performed some years before, and it was considered useless to repeat it and attempt relief in this manner. Comparatively large quantities of pus flowed from both frontal sinuses during each daily irrigation.

December 28 the patient complained of his pain returning, despite the relief which he seemed to have enjoyed from the frontal sinus irrigation. A right ethmoid operation was performed to relieve the drainage on that side, which was more or less impaired by a septum deviation. The patient seemed to be much improved.

On Jan. 4, 1913, a partial turbinectomy on the left side was performed and a radical intranasal for the relief of the antrum on this same side. The pus still continued to flow from the sphenoid and frontal on the right side, though less from the left frontal sinus. All this time, the patient was receiving inunctions

of potassium iodid and injections of the citrate of iron to combat the anemia from which he was suffering. The hemaglobin at the time he was received as a patient was recorded as less than 65 per cent.

The irrigation of the sphenoid and frontals was continued until February 1, 1913, when an attempt was made to operate on the left sphenoid. This attempt was frustrated more or less by the thickness of the bone and the inability of the operator to reach the floor with the rongeur. The cavity was quite large, being irregular in its outline. This made it difficult for the curette to do its work as thoroughly as intended.

Figure 1.

On April 22, 1913, a left ethmoid operation was performed and the patient improved remarkably, both in bodily and mental vigor, until the middle of the latter part of November, 1913. From April to the latter part of November, the patient had made frequent visits for irrigation of the frontal sinuses, which were still draining freely. Neither the antisyphilitic treatment, nor the autogenous vaccine, changed, at any time, the nature or the amount of the sinuses or bronchial secretions.

After considerable debating with the patient concerning the return of his frontal pain, he finally consented to an exploratory

operation of both sinuses, with the understanding that, if, in the judgment of the surgeons, radical operations were indicated, nothing would be done at this time other than curetting the cavities and keeping the drainage open.

To our great surprise, and though the Roentgen-rays did not show it, a large sequestrum, embracing the septum between the sinuses and the nasofrontal process, was removed and the surrounding bone was attacked freely down into the healthy portions of the frontal bone, both above and below. The use of bone wax was omitted. The drainage was made down into the nose with a rasp, and an external and internal drain put in situ, and the patient told the result of his operation.

He was urged to consent to a radical operation at once, despite the disfigurement which would take place, as the sinuses were deep and wide and the depressions left by the removal of the external walls would necessarily alter his appearanc to a certain extent.

The sinuses were irrigated and repacked twice daily for over three weeks, the patient hoping all this time that the condition would improve. This was not to be, however, for on December 25, an edematous condition developed over the frontal region, simultaneous with swelling in the left temporal region and an exophthalmos on the same side. We were then permitted to take the patient to the hospital with the understanding that we were to perform whatever operations might be necessary in our judgment. Assisted by Dr. G. A. Renz, his family surgeon, a radical operation (Knapp's) was performed on both sides on December 27. The erosion found in the outer walls and floor of the frontal sinuses was so marked that both anterior walls were removed in one piece with hardly any force. A subtemporal abscess opened externally, and a large sequestrum was found, including both tables and extending an inch and a half just above the site of the first sequestrum, including the septum. Every effort to reach all the diseased bone was made and the patient made an uneventful recovery, being much surprised at his slight disfigurement. The nose for the first time was free from any suppuration, and the patient was relieved by this improved condition.

Delight, however, was of short duration, for an February 19, a swelling in the left temporal region had taken place in the night and exophthalmos of the left eye was present. For the first time I dreaded what was before me, and though the incision and removal of a small sequestrum was followed by rapid improvement, the same thing occurred on the right side on February 24, resulting in evacuation of pus, and reoccurred on March 4 with exophthalmos. No eye changes had been noticed on either side up to this last time. The patient complained of a dull headache in the right frontal region, however, and the optic nerve this time showed considerable redness and some swelling. The patient, who had returned home, was taken to the hospital on the same day; a wide incision was made through the temporal muscle and careful search instituted for sequestration and a possible subdural

abscess in the anterior cranial fossa, both of which were found. The latter was drained and repacked for over twenty days. From this time on it was a continued search and lookout for fluctuation and sequestration. Dr. G. A. Renz from this on carefully watched the patient's general condition and was of great assistance to me in keeping his resistance to the highest level possible. A large sequestrum involving the frontal bone at its junction with the parietl bones over the longitudinal sinus, was removed March 28. A smaller one over the left center of the parietal bone was removed April 24.

Figure 2.

Dr. Todd, who saw this case in consultation with me at about this time, concurred with me in the opinion that utmost vigilance was an important factor in the treatment of the case.

Toward the middle of May, the patient's hearing began to fail. On May 18 a bilateral operation for mastoiditis was performed as quickly as possible, which brought a return of hearing and arrested drainage within a week. By this time the patient had had from nine to ten drains in the parietal and occipital regions, all of which were granulating and healing of their own accord under a wet bichlorid dressing, which had been applied as a constant dressing for many weeks with apparently good results and the

reformation of new bone in the frontal and other infected fields, as shown by the roentgenograms.

The patient, by this time, had lost some weight, but his vitality was remarkable, though his anemia was quite marked. On June 3, when told that all his wounds were healed and he could be discharged from the hospital, he performed several acrobatic feats in the dressing room. He preferred, however, to remain in the hospital a few days longer, and on the evening of the 7th he was taken with a severe pain over the lower apex of the left lung. An examination, a few hours later, revealed an area of dullness which rapidly increased and resolved itself into hypostatic pneumonia, from which he died June 9, 1914.

To all appearances, for more than two weeks up to the time of his death, the furor of the infection seemed to have reached its climax. The temperature, which had reached at times 102½, was then ranging from 99 to 100 in the afternoon, and outside of a tight feeling about the head, the patient was having absolutely no discomfort, and one would have scarcely realized the tedious siege he had been having for the past six months.

There is a probability that this man might ultimately have died of meningitis, as many have before him, or some other remote condition arising therefrom.

That the production of new bone was going on at a rapid pace may be seen from the pictures I here exhibit. Apparently the process had come to a standstill below the region of the trochlear.

My only regret is that the patient's pulmonary condition was such as to cause his death before time could demonstrate whether the many expedients which had been resorted to in order to bring about a cure had really been as successful as the clinical picture seemed to manifest. The lung condition had been realized as serious from the first, and attempts to relieve the patient of the discomfort from the copious discharge of the sinuses were, in a measure, an attempt to reduce sepsis to a minimum in a case in which a chronic purulent bronchitis was already so well established.

The writer fully realized the dangers of operating on a man suffering from such pathologic conditions, and felt that no attempt should be made which would, in our present knowledge, endanger the patient's life.

Cases such as the one I have attempted to report as fully as possible are of such rare occurrence that I hope this Society will feel at liberty to freely criticize and make suggestions which may alter the course of such rapid and general disintegration of

the bones of the skull through diploetic invasion in cases with this history.

In connection with this case, I wish to state that my first experience with the Ogston-Luc operations dates back some eighteen years. In a series of fourteen so-called atypical cases, published in 1914, in which tuberculosis, syphilis, or any other chronic systemic infection had been excluded, the writer was able to report fourteen cures without any complications of any kind, and firmly believes that the operation could be performed without danger, in healthy and particularly young subjects, if the pathology and bacteriology of said cases were thoroughly understood.

Fig. 3.—1. Sequestrum removed from vortex. 2. Parietal. 3. Frontal above right and left frontal sinus. 4. Right temporal. 5. Left temporal.

My reason for classifying these cases as atypical is because, as far as the patients were concerned, there was no reference made to the sinus, but only to the actual symptoms from which they were suffering, such as tic douloureaux, nausea, and dyspepsia, nocturnal epilepsy, vertigo, and dizziness, asthma, disturbance of vision, periodical pain in the eye, etc. The pictures of some of these I offer for your inspection in connection with the report of this case, although I expect to report each more fully in the future.

DISCUSSION

Dr. Joseph C. Beck, Chicago: This case ilustrates how hopelessly treatment of this disease may be in this region even with modern methods or, for that matter, even with the use of mercury. The essayist will remember that, while at the American Medical meeting at Minneapolis, I was asked to do a radical frontal sinus operation, and that in exploring I found a typical case of syphilitic osteomyelitis in that region extending

way over to the top of the head. As soon as I made the diagnosis I closed up the wound and recommended antiluetic treatment. It seems to me that in the case before us the salvarsan should not have been stopped with three doses, but that fifty doses should have been given if necessary, that is to say, until you got a negative Wassermann. Mercury itself and potassium iodin have left us many a time in the lurch, but that salvarsan will do the same in these cases I have my doubts. Certainly the doctor is to be congratulated because he got the patient along so far. He lost him by letting him do too much acrobating.

Dr. Albert H. Andrews, Chicago: I want to call attention to the relation between accessory sinus diseases, as was presented in this case, and tonsil disease and pneumonia. Investigations are going on to establish the relation, and the work that Dr. Seydell did on the tonsils, and this case and many other cases which we have seen, point very strongly to the tonsils and the accessory sinuses as the etiological factor in pneumonia, and that the infection does not have to reach the lung from a sinus thrombosis, as was formerly supposed. It is a subject which should be investigated, and I hope some one better qualified than I am will go on with it and get some results.

Dr. Harry Pollock, Chicago: I desire to report a case similar to that of Dr. Beaudoux', although not quite so extensive in its destruction. A man about 30 years of age, father of two healthy children, presented himself with a necrosis of the antrum extending well up into the frontal region. It was of about a year's standing when he came to us. He had already had the ethmoids curetted and the antrum opened. We obtained a negative Watermann. We gave him a provocative dose of salvarsan, and there was still no positive Watermann. We treated him expectantly, so to speak, but necrosis continued, and on three or four different occasions we removed several large sequestrums from this region, and finally we began to push our salvarsan. He received at least twenty doses of salvarsan, we giving them until we could find no more veins for injection. For the last two doses I used the external saphenous vein. His condition began to gradually improve, and after a period of a year and a half, he recovered. He never had a Wassermann positive, and there was nothing else clinically except a syphilitic necrosis.

I wish to mention the point brought out by Dr. Beck in regard to giving these patients salvarsan. There is no limit. This patient probably received the most we ever gave. We have given in these cases as high as ten and fifteen doses before seeing any definite results, and if we do give this large number of doses we will get results.

Dr. Carmody, Denver: One point I wish to make in regard to a case of my own. The patient was operated on twice by myself and given three doses of salvarsan, and then put on mercury and iodids, but he did not have the confidence in me that Dr. Beaudoux' patient had in him, so he went to a colleague, and the condition cleared up while he was taking potassium iodid. Notwithstanding, I believe the first doses of salvarsan were simply provocative doses.

Dr. Beaudoux (closing discussion): In regard to salvarsan, I am not positive, Dr. Beck, whether it was three or four doses, but it was not over that. I remember reading a short article stating that sometimes salvarsan increased the necrotic process, and this made us rather timid about continuing its use, so the patient was put on mercury and iodin until his anemia became quite marked. It could not be controlled with iodide of iron and other iron preparations, so we discontinued it after thirteen or fourteen months. As to the teeth, these were well looked

after. In this case, we had a mixed infection with a much lowered resistance. His opsonic index was as high as he could possibly get it and his leukocytosis was overdoing itself.

In cases which present a chronic systemic dyscrasia of long standing, particularly syphilitic or tuberculous, it is not of particular advantage, and very dangerous, to do sinus or nose surgery unless one is compelled to, as I was in this case. At first we did only what we thought was safe to relieve the man of his offensive discharge, just as he had been treated by some of my colleagues. I do not think in a case of this kind that it would have been wise to push the mercurial medication to any further extent than we did. Consultants did not advise it.

It is to be regretted that, after going through so many serious complications and operations, his pneumonic complication could not have been postponed to test the efficacy of our surgical work and endeavors.

THE TONSILS AS AN ATRIUM OF INFECTION IN POLIOMYELITIS

ERNEST M. SEYDELL, M.D.
WICHITA, KAN.

The recent reports of Mathers, Nuzum, and Rosenow on the etiology of anterior poliomyelitis, which seem to show definitely that the causative organism is not a filterable, ultramicroscopic virus, as previously considered by Landsteiner and Levaditi, and Flexner and Lewis, but very probably a micrococcus, have opened some new avenues for investigation. Since it has been shown in a number of cases that the same organism found in the spinal fluid and tissues of the brain and cord has also been found in the tonsils of these same patients, it naturally makes one ask the question: Are the tonsils the portals of infection? Anterior poliomyelitis is an infectious disease, accompanied in a certain proportion of cases by paralysis. The percentage of cases in which paralysis occurs is a great deal lower than has been previously believed; perhaps one in four.

BACTERIOLOGY

In 1905 Geirsvold,[1] in a preliminary communication, stated that he had successfully isolated from the cerebrospinal fluid in twelve acute cases of poliomyelitis a gram positive coccus, occurring in pairs, short chains, and tetrads. He stated that he was able to make cultures of this micro-organism on a variety of medium, and further, that he was able to produce definite paralysis in white mice, rabbits, and pigeons. Landsteiner and Popper[2] and, working independently, Flexner and Lewis[3] determined that the virus of poliomyelitis passes through a Berkefeld filter, and were able to produce a disease in the monkey which, in its clinical, pathologic, and histologic features, was quite similar to acute poliomyelitis in man. They were unable to produce the disease in mice, rats, guinea-pigs, dogs, cats, goats, sheep, or horses. In 1913 Flexner and Noguchi[4] cultivated and demonstrated, microscopically, a small filterable micro-organism

1. Geirsvold, M.: Norsk Mag. f. Lægevidensk., 1905, **3**, 1280.
2. Landsteiner and Popper: Uebertragung der Poliomyelitis acuta auf Affen, Ztschr. f. Immunitätsforsch. u. exper. Therap., 1909.
3. Flexner and Noguchi: Jour. Exper. Med., 1913, **18**,, 461.
4. Flexner, Simon, and Lewis, P. A.: Nature of the Virus of Epidemic Poliomyelitis, Jour. Exper. Med., 1913, **18**.

with which they produced poliomyelitis in monkeys. The cultural conditions were similar to the bacteria; but they did not classify the micro-organism, rather inclining to the view that the disease was caused by a virus. In September of this year, Mathers[5] published a report in which he stated that he had isolated a gram positive micrococcus from the brain and cord of fatal cases ocurring in the Chicago epidemic. He obtained these organisms in six out of seven cases examined. He did not obtain the micrococcus from the spinal fluid or heart blood. In closing, he stated that in view of the accepted fact in regard to the filterable virus of epidemic poliomyelitis, it would seem most reasonable to regard the micrococcus as a secondary invader. A short time after this Nuzum[6] and Rosenow,[7] working independently, Nuzum in Chicago, Rosenow in New York, published the results of their observations, both having undoubtedly discovered the same organism. Nuzum found the organism in each of the twelve cases examined by him. The micrococcus was obtained from the brain and cord at necropsy. He also reported the finding of this same micrococcus in the spinal fluid during life in eight of nine cases examined. Since then these examinations have been extended, and in a subsequent report[8] he adds that he has found the same organism in forty-five out of fifty cases, or in 90 per cent. Twice he cultivated the micrococcus from the heart blood of the patient during life. The organism has been found in the spinal fluid twenty-four hours after the onset of the disease, and may still be found there two months after the patient has apparently recovered. Rosenow first, and later Nuzum, demonstrated these organisms in the tonsils of patients suffering with poliomyelitis. The former also found them in the ventricular fluid after death, in the blood during life in one instance, but not in the spinal fluid. He also isolated the organism from the brain and cord in each of the twelve cases examined.

The micrococcus is polymorphous. It appears in a minute form on anaerobic cultivation and readily grows out into a larger form when cultivated aerobically. It is usually found in short chains and clumps and also in diplococcus arrangement. The

5. Mathers, G.: Some bacteriological Observations on Epidemic Poliomyelitis, The Jour. Am. Med. Assn., Sept. 30, 1916.

6. Experimental Studies in the Etiology of Acute Epidemic Poliomyelitis, The Jour. Am. Med. Assn., Oct. 31, 1916.

7. The Etiology of Epidemic Poliomyelitis: The Jour. Am. Med. Assn., Oct. 31, 1916.

8. Bacteriological Findings in Cerebrospinal Fluid in Poliomyelitis, Jour. Am. Med. Assn., Nov. 11, 1916.

smaller forms can be passed through a Berkefeld filter, and the subsequent cultures show both the large and small forms of the organism.

ANIMAL EXPERIMENTS

In 1913, Flexner and Noguchi produced poliomyelitis in monkeys, with their filterable virus. Rosenow produced paralysis with lesions in the central nervous system in guinea-pigs, rabbits, dogs, cats, and monkeys by intravenous and intracerebral injections. He produced the same lesions by injecting emulsions of pus expressed from the tonsils; also emulsions of the extirpated tonsils and brain tissues.

Nuzum made cultures from the tonsil crypts in eleven cases of acute poliomyelitis and from eight pairs of tonsils removed from patients suffering with the same disease,[9] and from all of the above, with the exception of two cases, the same peculiar gram positive micro-organism was isolated repeatedly, not in pure culture as a rule, however. With these cultures he produced paralysis, etc., in fourteen out of seventeen rabbits inoculated. In one *Macacus rhesus* monkey and small ring-tail monkey injected with the tonsil cultures, there have been no results to date. Positive results were obtained in rabbits with cultures from the nasal discharge of two cases, from emulsions of brain tissue in two others, and also in one case using mesenteric lymph gland emulsions. The monkeys developed the typical lesions when injected with cultures obtained from the spinal fluid.

The rule that the younger animals are more susceptible to inoculation holds true as it does in the human race. It may be said that the incubation period varies from thirty hours to fourteen days; also that the picture produced in the monkey is typical of acute poliomyelitis, both clinically and pathologically. In young rabbits there is a variation in the microscopic picture, although many of the changes attendant on the disease in man and monkeys are present in the central nervous system after paralysis.

THE ATRIUM OF THE INFECTION

Now arises the question: Where do these germs enter the system? I might say that it is impossible to definitely answer this most important query at this time, but believe from the evidence already accumulated that the tonsils play an important

9. The tonsils were removed by Dr. Karl A. Meyer of the Cook County Hospital.

rôle in this direction. It has been the supposition for a number of years that the infection is disseminated from the nasopharynx of the diseased to the nasopharynx of the unfortunate victim. In America, Sohier Bryant,[10] Stephens,[11] Anderson and Frost, as quoted by Bryant, Sheffield,[12] and others, state that in many cases acute poliomyelitis is preceded or accompanied by nasopharyngeal inflammation, and suggest that many cases presenting a nasopharyngitis during an epidemic may be an abortive type of this disease. Experimental work should now be undertaken to prove the truth of these assertions. Rosenow[13] states that the tonsils in patients suffering with poliomyelitis, especially those over 3 or 4 years old, yield on pressure a surprisingly large amount of infectious material, even when no objective symptoms of tonsillitis are present.

These tonsils present numerous areas of localized necrosis, usually along the capsule and not communicating with the surface. They contain a peculiar opalescent material in which are found large numbers of the micrococci. These have also been found in the adenoids. In approximately twenty-five cases of infantile paralysis in which the temperature remained high and the paralysis was progressing, tonsillectomy was performed. No deleterious effects were observed in any of these cases; on the contrary, it was found that following tonsillectomy the temperature abated and convalescence was rapid. The results were so striking in some of the above cases that Dr. Rosenow suggests the advisability of removing the tonsils in this type of cases.

The next question which naturally arises at this place is: How many of the patients that have had poliomyelitis had previously had their tonsils removed? To help determine this important question, Dr. Meyer (Cook County Hospital) and the writer have carefully analyzed the histories of over 200 cases which were treated at the Cook County Hospital during the recent epidemic. Our findings may be tabulated as follows:

Total number of cases studied, 203.

Average age was 4 years.

Oldest patient suffering with the disease was 50 years of age.

Youngest patient was 6 weeks old.

---

10. Epidemic Poliomyelitis; Its Nasopharyngeal Aspects, New York Jour. Med., October, 1446.
11. Stevens: Intercolonial Medical Journal, 1908.
12. Sheffield: Med. Rec., August, 1916.
13. Rosenow: Discussion of R. N. Lovett's paper, New York Med. Jour., Oct. 14, 1916.

Deaths in this series numbered ten, or 5 per cent.

Average age of fatal cases was 4½ years.

Oldest patient to die was 26 years.

Youngest was 10 months.

In this number, twenty-one cases, or 10 per cent., had some cervical adenopathy.

Fifteen cases, or 7½ per cent., had an angina as an initial symptom.

Ten cases, or 5 per cent., had some discharge from the nose.

I might state that the Chicago Board of Health figured the mortality at 21 per cent. This was reduced in this series by the fact that some of the cases diagnosed as poliomyelitis proved at postmortem to be tuberculous meningitis or brain tumor.

In the 203 cases whose histories we examined, we find that in the majority the tonsils were recorded as small. In a very much smaller number they were stated to be enlarged and slightly congested. In two cases one tonsil had been removed, and in one other a tonsillectomy had supposedly been performed. On examination, however, I found some tonsil tissue on the right side which contained a large crypt and found also an adenoid. In these cases in which a portion of the tonsil tissue had been removed, the paralysis was slight and the convalescence rapid. In a communication from Dr. Rosenow on this subject, he states: "I know of two cases that have developed poliomyelitis in whom tonsils and adenoids were presumably properly removed. In one, there was only a slight weakness of the shoulder muscles, which disappeared promptly. In the other case, the paralysis was slight, even though a brother of the patient, whose tonsils had not been removed, was severely paralyzed. I have seen these two cases, and have heard of a few others, and I believe in all cases the paralysis was not marked."

CONCLUSION

A review of the findings as shown by the Chicago epidemic does not lead one to believe that many of the cases had a nasopharyngitis, either as an initial symptom or later in the disease. It must be remembered, however, that many of these cases were brought into the hospital with the disease well advanced, and it was sometimes very difficult to obtain any definite history. In this epidemic the organism did not produce cervical adenopathy, neither did it seem to produce apparent inflammation of the

mucous membrane covering the tonsils, as in most instances in which abscesses were found the tonsils appeared normal.

Three investigators working independently in widely separated cities have recently isolated what appears to be the same peculiar micro-organism from the tonsils, tonsil crypts, spinal fluid, brain and cord, and even from the heart blood and mesenteric lymph glands of patients affected with poliomyelitis. Their experimental reports indicate that the micrococcus has met all of the provisions of Koch's laws.

It has been known for a long time that the causative factors of poliomyelitis remain in the nasopharynx of the patient for as long as six months. Now we find that we can produce the disease in animals by injecting them with the contents of abscesses found in the tonsils of the diseased patient. The disease attacks young children in a very high percentage of the cases, who, as we well know, have a tendency toward abnormal conditions in their lymphatic systems. One of the striking features of this disease is the elective affinities of this organism for the nervous system on the one hand, and for the lymphatic system on the other. The germs seem to travel to the central nervous system along the perineural lymph channels.

In the 203 cases which we studied, we find tonsils present in 200 cases. In three there was a partial absence of these structures, and in these cases the paralysis was slight and recovery rapid. In the cases of Rosenow we find a similar course and result.

All of the above data very greatly strengthen our belief that the tonsils and adenoids are the portals of infection in this alarming disease.

In closing I wish to thank Dr. Nuzum and Dr. Meyer, who made it possible for me to publish the reports of the Cook County Hospital.

DISCUSSION

Dr. Joseph C. Beck, Chicago: This paper illustrates many, many things. First, the importance that this subject has acquired from the wide publicity that this disease has been given in the public press during the past year. When this paper becomes known and is published in the press, as usually happens, it will mean that in every community in which there has been an epidemic, there will naturally be a cry for the prevention of this disease, and a demand that the tonsils be removed from every child. If what has been stated by Dr. Seydell can be definitely proven, the demand will have to be met. The staff of Cook County Hospital is greatly indebted to Dr. Seydell for going there and doing this work. Another point is shown, namely, that men in the smaller cities need not complain that they do not have a clinic, because Dr. Seydell has demon-

strated that men go away from home and do good work. I saw some of these patients after they were operated on. There is not much to be said by way of discussion, but one thing I would call attention to and that is the length of time these micro-organisms remained in the tonsils after the patients recovered from the disease. They have been known to stay in the nasopharynx for six months. The tonsils of these patients are carriers of infection, and if the tonsil is the atrium of infection, then the rest of the infectious material in the body is a very important point.

I wish to mention, too, that the infection travels by way of the perineural lymphatics. We know from Dr. Killian and Dr. Magruder that the sinuses are probably a great source of infection; that through the sinuses is the great path for perinueral lymphatic infection, and it is from there the germs pass through to the cerebrospinal fluid. I wish some one would do some work along that line in connection with this disease, and see how much the sinuses are infected.

Dr. J. A. Pratt, Aurora, Ill.: I want to report one case from our town last summer. One of our families visited in Boston. They had four children; the oldest was 9 and the youngest was 2 years. After returning to Aurora, the youngest child was taken with infantile paralysis, with the result that one leg is badly paralyzed. The other three children, who had previously had their tonsils and adenoids removed, did not contract the disease. The youngest child had not had them removed, which would seem to add weight to this theory.

Dr. Seydell (closing discussion): I have very little more to say. You understand, of course, that one attack affords immunity. I believe the average age, if you leave out a few of the older patients, one of whom was 54, another 50, and two or three over 30, is about 2 or 2½ years. Another point is, that it is not often the paralytic cases that spread the disease. It is the abortive cases, and something must be done to clean up these carriers. The germs are probably in the tonsils and in the adenoid tissue of the nasopharynx. The sinuses in these cases were not examined. Only two of all those reported had a purulent discharge from their nostrils. In these cases it was of greenish hue and on inoculation produced the disease in rabbits.

THE TECHNIC IN TONSIL OPERATION

AUSTIN A. HAYDEN, M.D.
CHICAGO

In presenting this paper, the author is so fully aware of the triteness of the subject that a foreword of apology seems necessary.

There are in the United States today probably about one million people who have had their tonsils removed according to one method or another, and for one cause or another; and it appears from the present general trend of medical opinion that this work has only just begun. The fact, too, that tonsillectomies have proven so efficacious in the prevention and relief of rheumatism, neuritis, nephritis, infections of the gall-bladder, appendix, stomach ulcers, myocarditis and endocarditis and, most recently, in infantile paralysis, seems to predict that the surgery of the throat and mouth will play an even more important rôle in medicine in the immediate future.

The author claims but little of the operative technic about to be described as his own. It is simply a narration in considerable detail of a method of tonsil evisceration that has been elaborated according to well defined surgical principles, and that has proven satisfactory in an experience of over 7,000 cases operated on during the past ten years. The manner of controlling bleeding is original, although other somewhat similar methods of suturing the pharyngeal pillars have been described. Simplicity has been the keynote in the instrumentation throughout, the ether-suction machine being the only expensive or complicated apparatus used.

From an anatomic standpoint, as far as the scope of this paper is concerned, the tonsil presents for description a superior and an inferior pole; a mesial, mouth or throat surface; a lateral or neck surface; and an anterior and posterior surface. The latter two lie under the anterior and posterior pillars, respectively, and together with the neck surface, are capsulated. The mouth surface is bare and through it the crypts empty into the throat (Fig. 11).

For general anesthesia, ether is invariably employed by the drop method. As soon as the jaw becomes moderately relaxed and the pharyngeal reflexes fairly well abolished, the suction machine is started, and almost every case is completed without reapplication of the mask.

For local anesthesia, 1 drop of adrenalin chlorid in 1 ounce of a 1 per cent. solution of novocain or 0.1 per cent. of cocain is used. Two injections are made into the anterior pillar, one high and one low; two into the posterior pillar, in the same relative positions, and two through the tonsil tissue just beyond the capsule, between it and the superior constrictor of the pharynx, one high up and the other low down. The pillar injections should be made into the mucous membrane, but not through it, and in such a way that blebs will appear on its surface (Fig. 11). In this way, almost complete anesthesia and a very satisfactory degree of ischemia is readily obtained. Whether cocain or novocain be

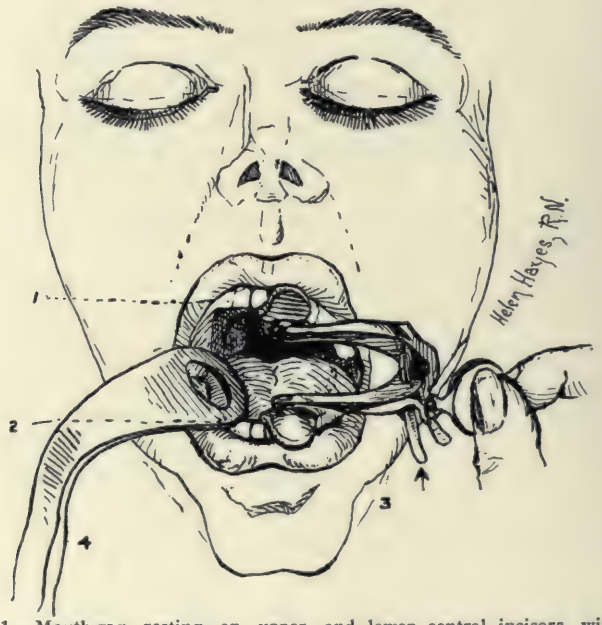


Fig. 1.—Mouth-gag resting on upper and lower central incisors, with tongue depressor prying jaws apart (general anesthesia). 1, upper incisors; 2, lower incisors; 3, mouth gag.

used makes little difference in the writer's opinion, provided the solution is of these strengths.

All children under 8 years of age should be narcotized. Above that age, the individual temperament of the patient should be the deciding factor in the choice of the anesthetic. The ether-suction apparatus makes it possible to work with the adult patient under ether fully as easily as if he were awake. More than half of the author's adult cases have been done under local anesthesia.

To lessen the salivary secretions, all patients (adults as well as children) get a small dose of atropin hypodermatically thirty

minutes before going to the operating room. Adults are given, in addition, an appropriate dose of morphin at the same time.

In using general anesthesia, the patient lies flat on the back without a pillow. Extension of the head over the end of the table is entirely unnecessary. The operator stands to the right, the assistant, nurse and instrument table are to the left, and the

Fig. 2.—Areas of injection into pharyngeal pillars and tonsil fossa. 1 and 2, anterior pillar injection—high and low; 3 and 4, posterior pillar injection—high and low; 5 and 6, tonsil fossa injection—high and low, showing lines of direction of hypodermic needle through tonsil.

anesthetist with the ether-suction machine is at the end of the table where she can steady the patient's head.

The mouth is opened by the tongue depressor, held in the surgeon's left hand and introduced between the teeth on the right side. The jaws are pried apart and the gag inserted between the upper and lower central incisors from the left side. Careful

manipulation here will avoid dislodgment of the teeth, which is of great importance in patients over 7 years. The ether tube is held hooked into the right angle of the mouth by the anesthetist. The suction tube is always placed well back in the throat on the side not being operated on, either in the empty tonsil fossa, or directly against the posterior pharyngeal wall (Fig. 1).

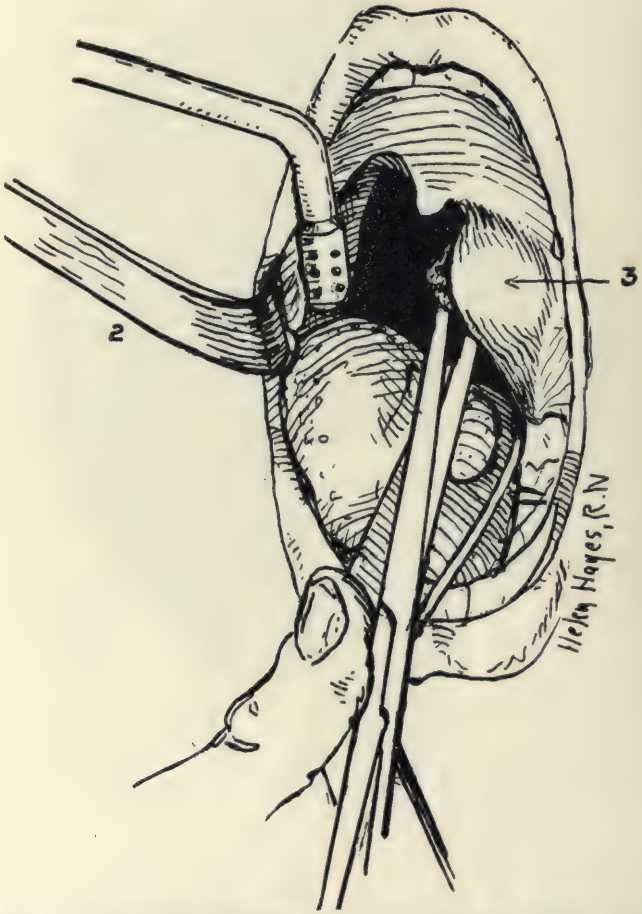


Fig. 3.—Tonsil outlining itself beneath the anterior pharyngeal pillar as it appears when drawn straight forward. Forceps grasping superior and inferior pole of tonsil. 1, suction tube; 2, ether tube; 3, anterior pillar.

If the case is being handled under local anesthesia, the patient is seated in an ordinary chair, properly robed and holding a towel in his hands, into which he is directed to spit. A nurse steadies his head from behind. The operator sits in front of and to the patient's left side within easy reach of the instrument table, behind

which the nurse takes her position. The assistant is seated to the surgeon's left.

The right tonsil is removed first. It is firmly grasped with the volsellum forceps near its upper and lower poles and drawn straight forward and not toward the median line, as has been so often described. In this way it outlines itself clearly underneath the anterior pillar (Fig. 3).

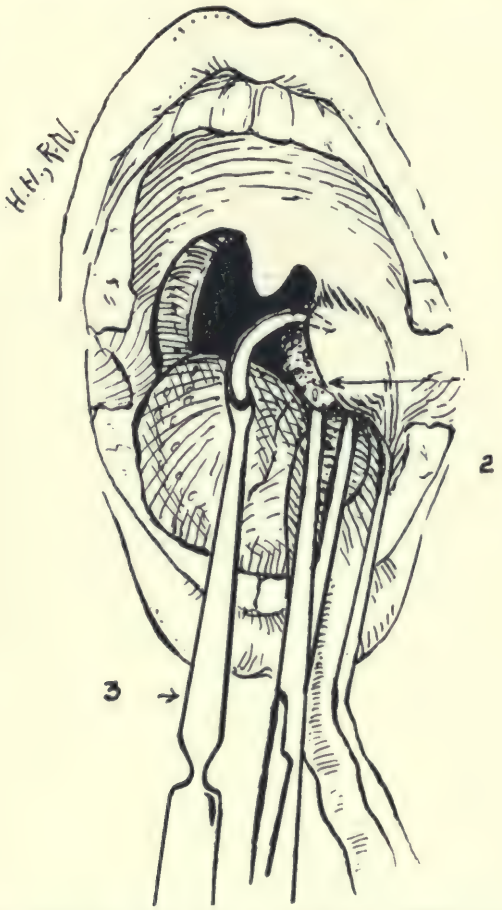


Fig. 4.—Anterior pillar knife under edge of pillar at superior pole of tonsil finding line of cleavage, pillar and capsule. 1, superior pole of tonsil; 2, margin of anterior pillar; 3, anterior pillar knife.

The anterior pillar knife is then introduced under the edge of the pillar at the superior pole, where it readily finds the line of cleavage between the tonsil-capsule and pillar. It then passes from above downward, always in plain view underneath the pillar, toward the inferior pole. Here the point is turned directly inward

toward the tongue, so that the part of the pillar which is attached to the lower jaw and base of the tongue remains intact. The glistening, white anterior surface of the tonsil is now readily exposed by pushing the freed, anterior pillar back with the blunt convex surface of the knife (Figs. 4, 5, and 6).

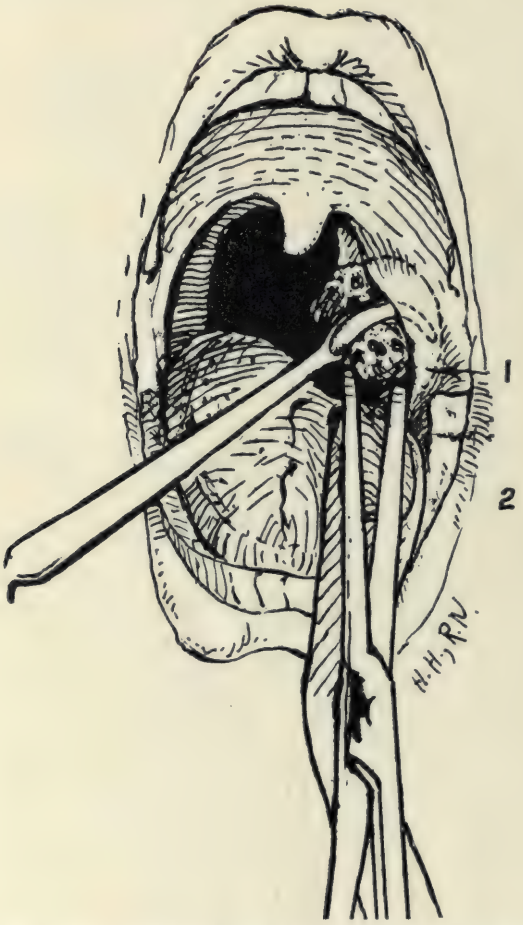


Fig. 5.—Anterior pillar knife going down in line of cleavage toward base of tongue. 1, anterior pillar attachment to lower jaw; 2, anterior pillar attachment to base of tongue.

The posterior pillar knife is now passed over the superior pole of the tonsil into the supratonsillar space and backward into the line of cleavage between the posterior pillar and the tonsil-capsule. It is then carried downward until the posterior surface of the gland is entirely free (Figs. 7 and 8).

The superior pole is now tilted inward toward the base of the tongue on the volsellum as far as the attachments of its neck surface to the fossa will allow. These attachments are kneaded away by the convex blunt surface of the posterior pillar knife, well down toward the inferior pole (Fig. 9).

The snare loop is now slipped over the forceps and made to encircle the remaining attachment of the gland to the lower part

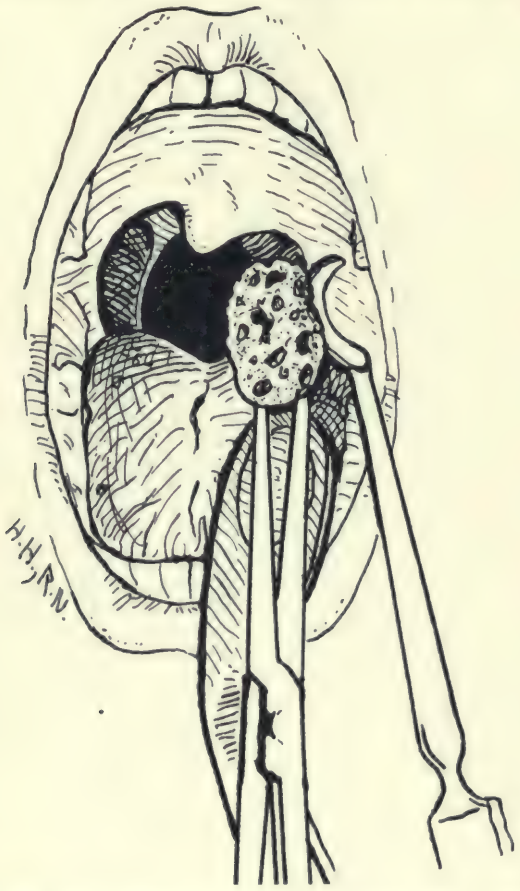


Fig. 6.—Anterior pillar attachment completely freed from pillar. Blunt edge of anterior pillar knife used as pillar extractor.

of the fossa, including the plica triangularis. Considerable care should be taken to keep the wire snugly against the tonsil base behind, with the slack of the loop in front. In this way the uvula is never in danger. The tonsil is removed by slow ecraseur.

The entire operation should not consume more than five minutes. If the suction apparatus has been used (general anesthesia),

it is well to notice whether or not the blood in the bottle has clotted. A fairly accurate idea of the patient's coagulation-period can thus be obtained.

The tonsil fossae of every tonsillectomy case should be dry when the patient is returned to bed. To allow a severed vessel

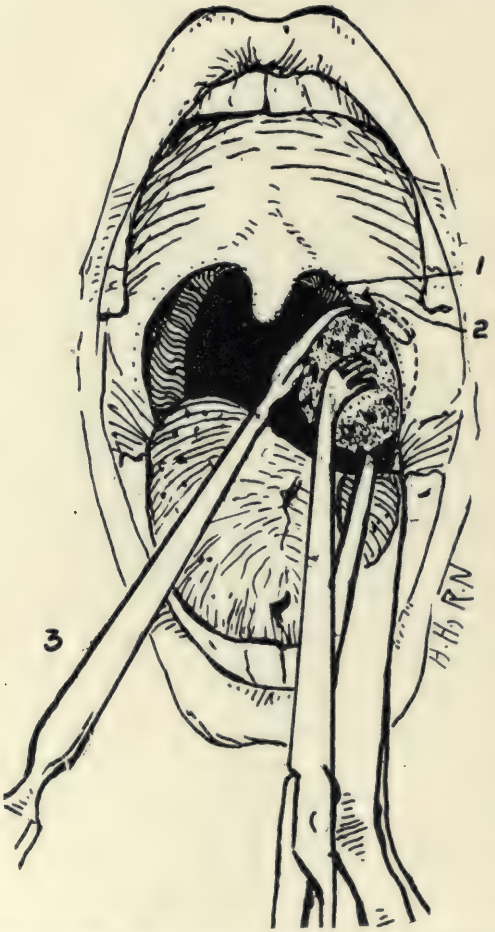


Fig. 7.—Posterior pillar knife behind superior pole of tonsil ready to commence separation of posterior pillar along its line of cleavage. 1, posterior pillar; 2, superior pole of tonsil; 3, posterior pillar knife.

to go untied is universally admitted to be bad surgery anywhere except in the throat. Here hemostasis has been left almost entirely in the hands of fate. Just why this should be so, is not apparent, especially when bleeding can be controlled with the same ease and precision, as in any other part of the body.

To do this, the anterior pillar must be drawn forward as soon as the tonsil has been removed, and the entire fossa explored for bleeding points. As all of the vessels that enter the tonsil are of macroscopic size, it follows that there can be no generalized oozing of blood in this locality, the frequent allusions to and the descriptions of the same to the contrary notwithstanding (Fig. 12).

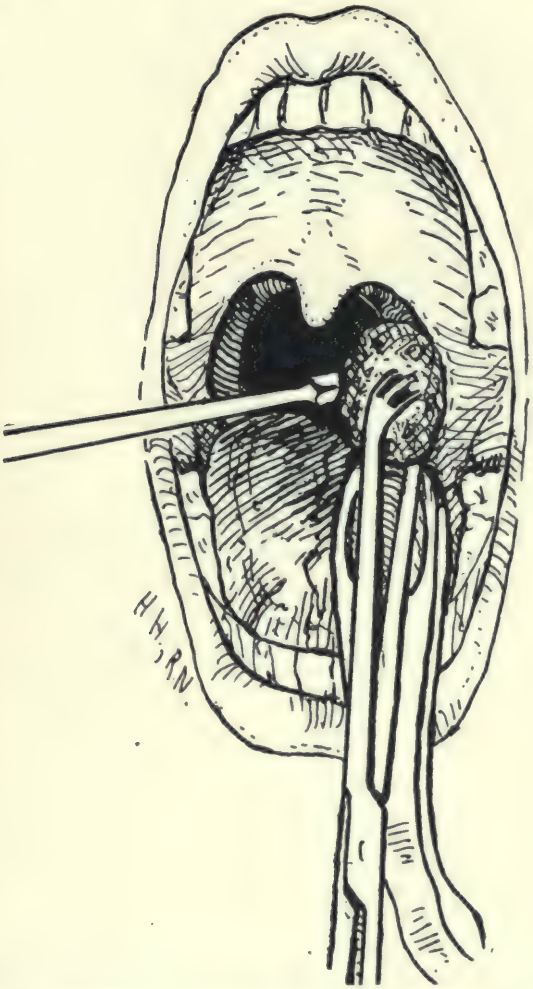


Fig. 8.—Separation of posterior pillar nearly completed. Posterior pillar knife behind tonsil.

Very frequently a vessel is found lying bare in the fossa a quarter of an inch or more. If its lumen is pointed against the smooth wall of the fossa, it can very easily give the appearance

of an indefinite general oozing, in much the same way that water coming out of a garden hose, whose end is being held directly against the ground, seems to well up all over, instead of spurting from one particular spot.

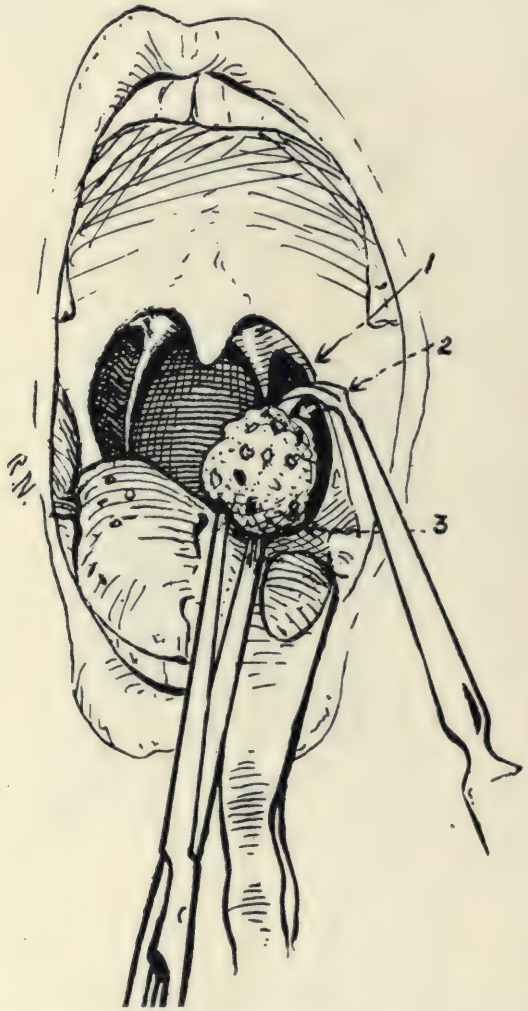


Fig. 9.—Separation of posterior pillar completed. Posterior pillar knife kneading neck-surface of tonsil from its fossa low down toward its inferior pole. 1, supra-tonsillar space (empty); 2, superior pole tonsil fossa (empty); 3, inferior pole.

On close examination, however, blood will be found to issue from a definite point. This should be caught immediately with a suitable artery-clamp, while the other tonsil is being removed; or, if the bleeding occur in connection with the second tonsil, the

hemostat should remain attached for a minute or so. If the bleeding still continues when it is removed, or if it was particularly active to begin with, or if, for any extraneous reason, such as

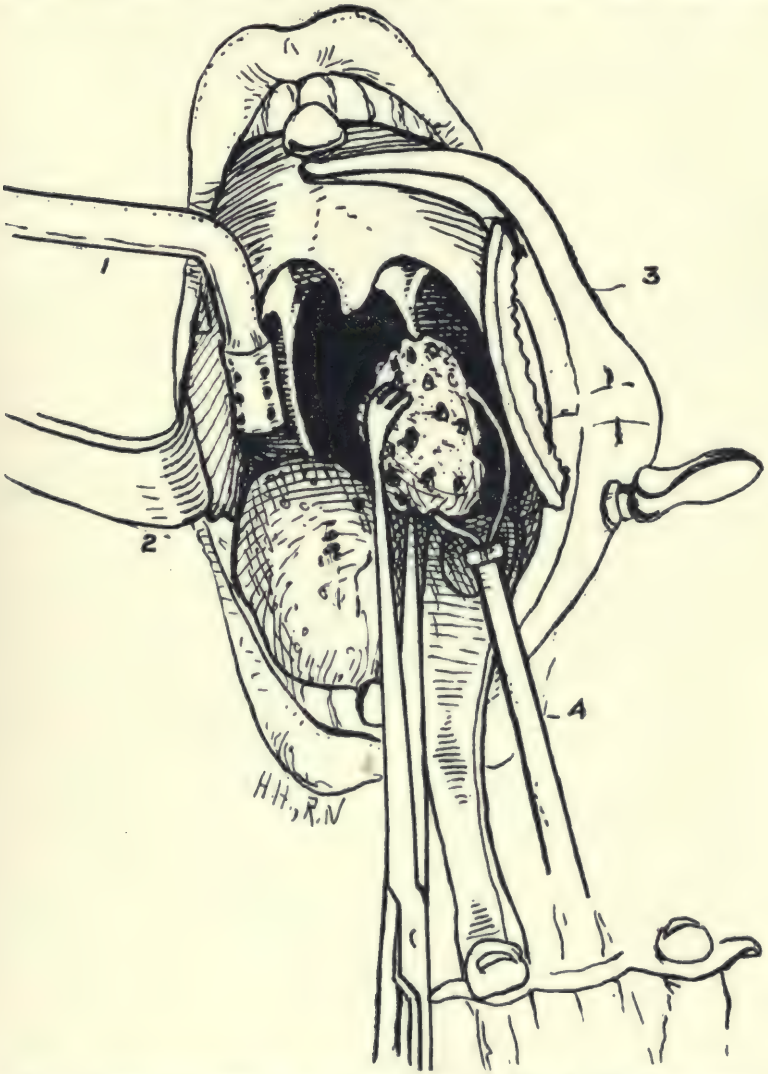
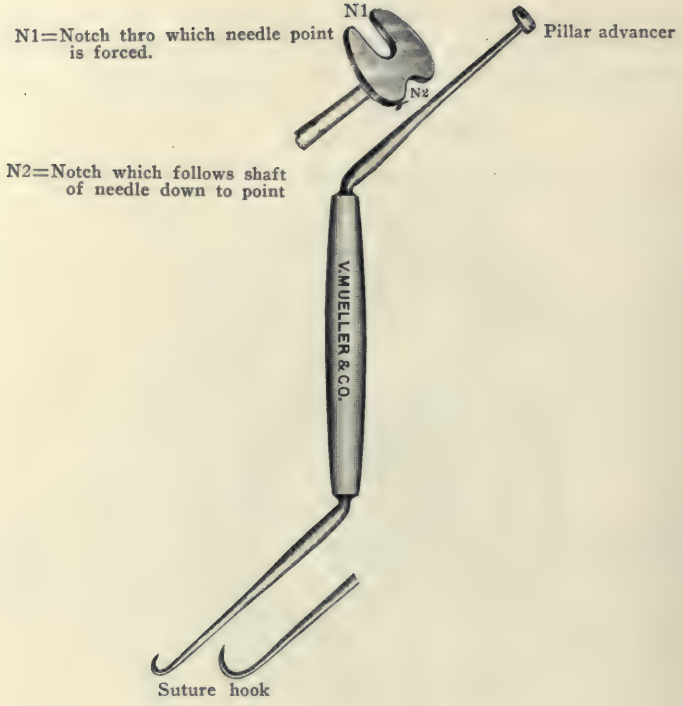
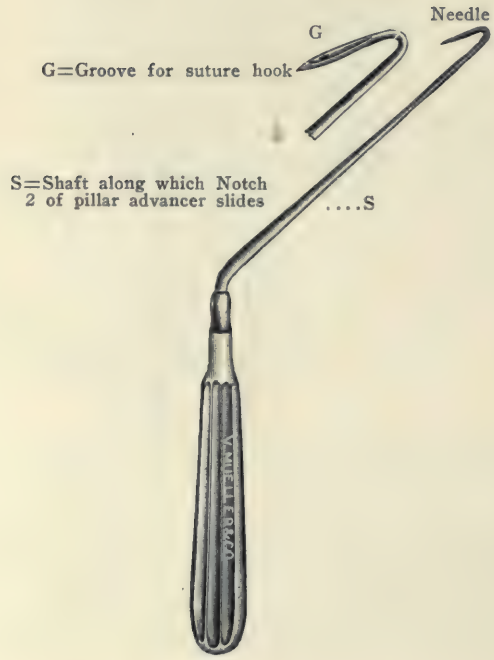


Fig. 10.—Snare in position. Slack part of wire in front of tonsil—not behind—to avoid engagement of uvula. 1, suction tube; 2, ether tube; 3, mouth gag; 4, snare in position.

high blood pressure, slow clotting, etc., troublesome hemorrhage is to be expected, the pillars should be immediately sutured.

To do this with the ordinary needle holder and the curved needle is a tedious, mutilating procedure. With the author's


N1=Notch thro which needle point is forced.
N1
N2
Pillar advancer
N2=Notch which follows shaft of needle down to point
V.MUELLER & CO.
Suture hook


Needle
G
G=Groove for suture hook
S=Shaft along which Notch 2 of pillar advancer slides
....S
V.MUELLER & CO.

instruments it is easy and almost painless, even though the operation be under local anesthesia. Iron dyed silkworm is used for two reasons: Silkworm ties more readily, and, everything considered, is much easier to handle in this very slippery locality than any other suture material. The iron dye color makes the stitch easily visible at all times.

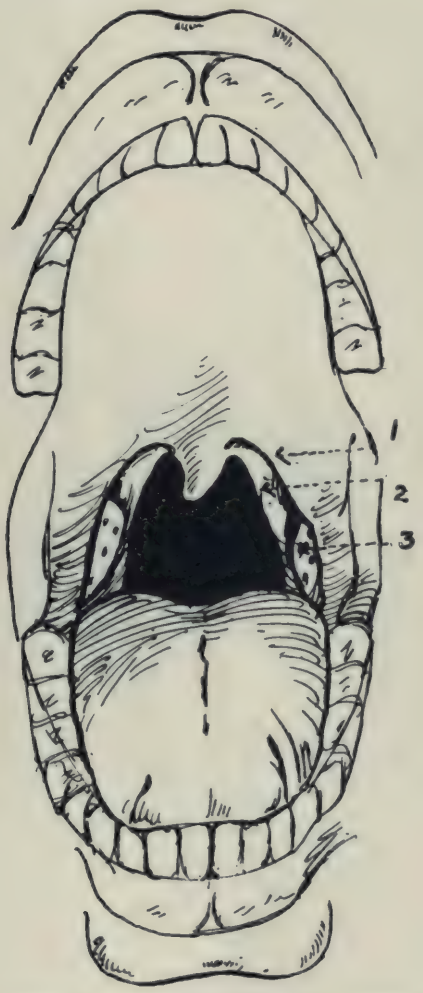


Fig. 11.—View showing location of small tonsils. 1, anterior pillar; 2, posterior pillar; 3, tonsil.

The soft palate is drawn forward; the threaded needle is introduced behind the posterior pillar opposite the bleeding point and passed deeply through it, and on superficially beneath the floor of the tonsil fossa toward the anterior pillar (Fig. 13). Here the

needle must stop, else the tissues, through which it is passed, because of their extreme friability, will be torn. The pillar pusher is now slid down along the shaft of the needle, whose point will

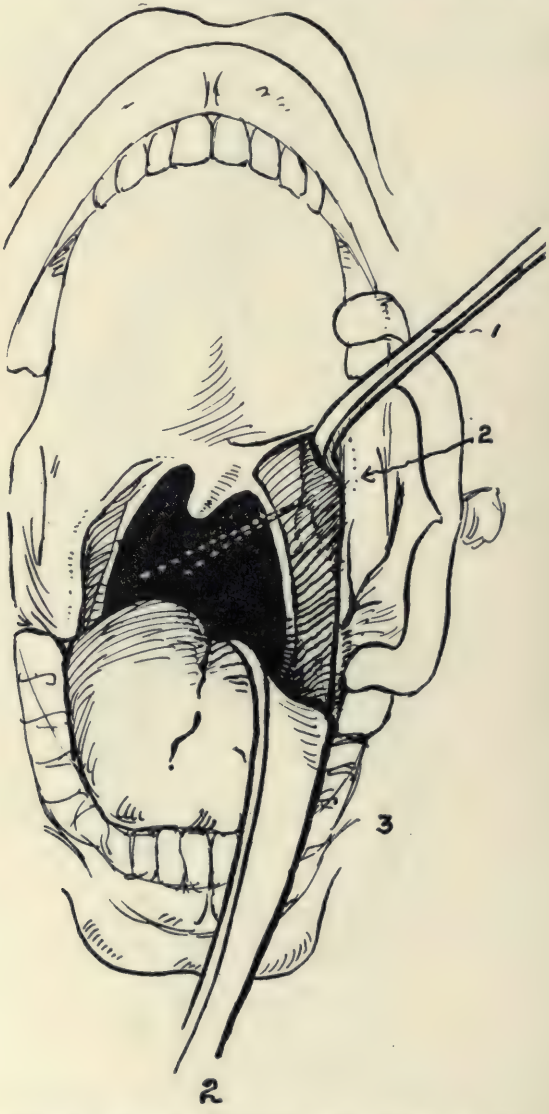


Fig. 12.—Examining fossa for hemorrhage after removal of tonsil. 1, pillar retractor; 2, bleeding vessel; 3, tongue held from side being operated on.

always come into the V-shaped opening, if the two instruments are kept parallel to each other. The pusher works the point through the anterior pillar until the eye, carrying the silkworm

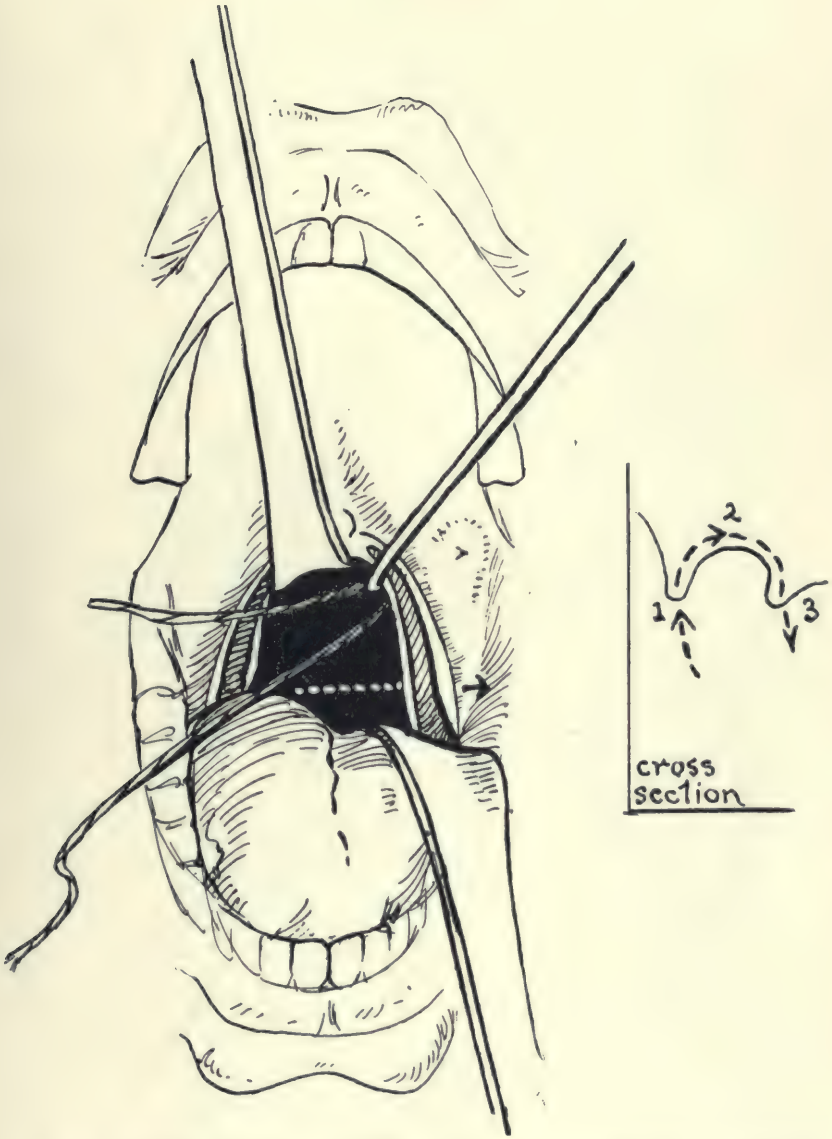


Fig. 13.—Needle piercing (1) posterior pillar, (2) floor of fossa and (3) entering anterior pillar. Also direction for another suture.

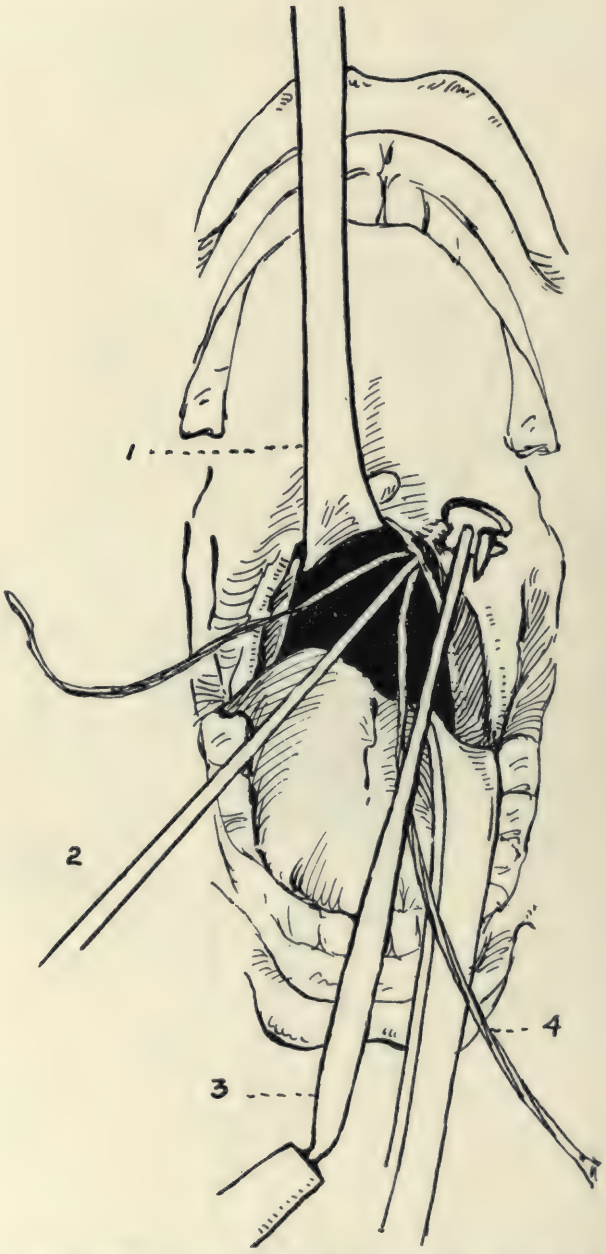


Fig. 14.—Using pillar-pusher to press needle point through anterior pillar. 1, soft palate retractor; 2, needle; 3, pillar pillar-pusher advancer; 4, iron dyed silkworm suture.

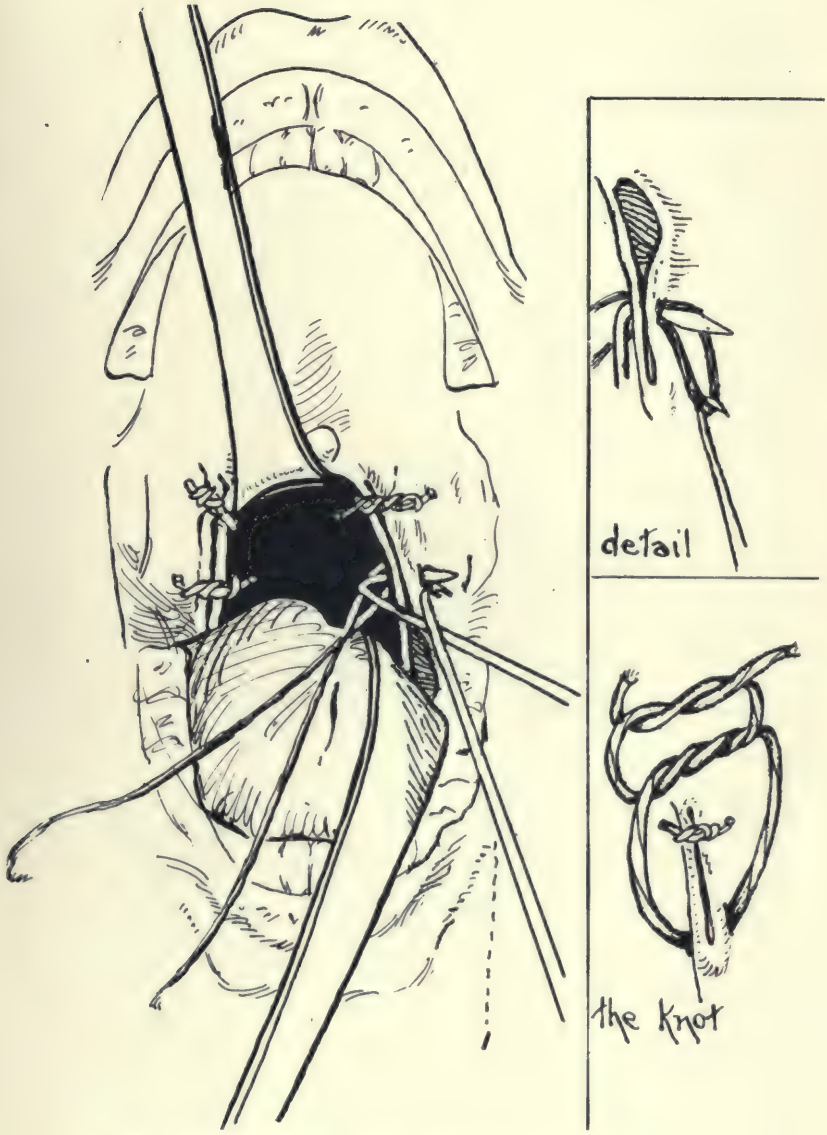


Fig. 15.—Pulling suture through with hook. 1, suture hook.

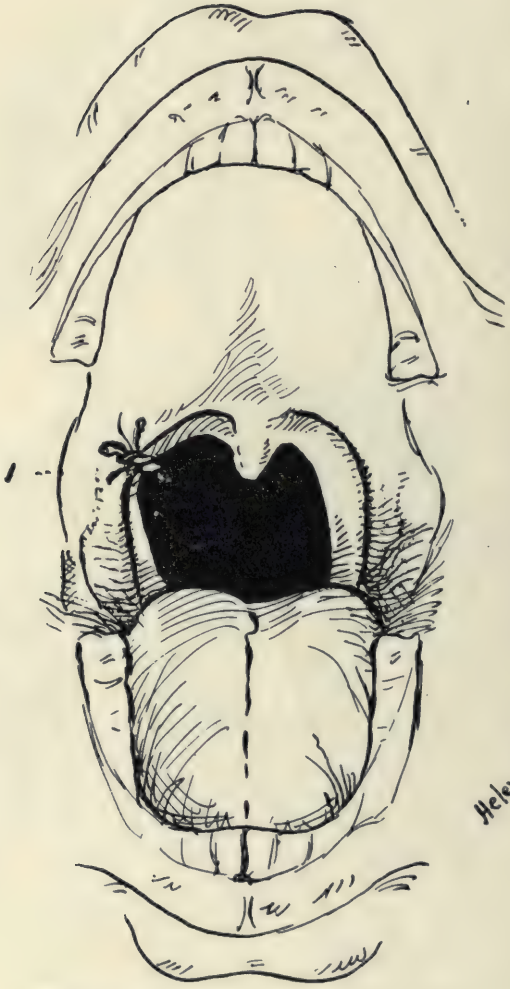


Fig. 16.—Suturing completed. 1, suture in situ.

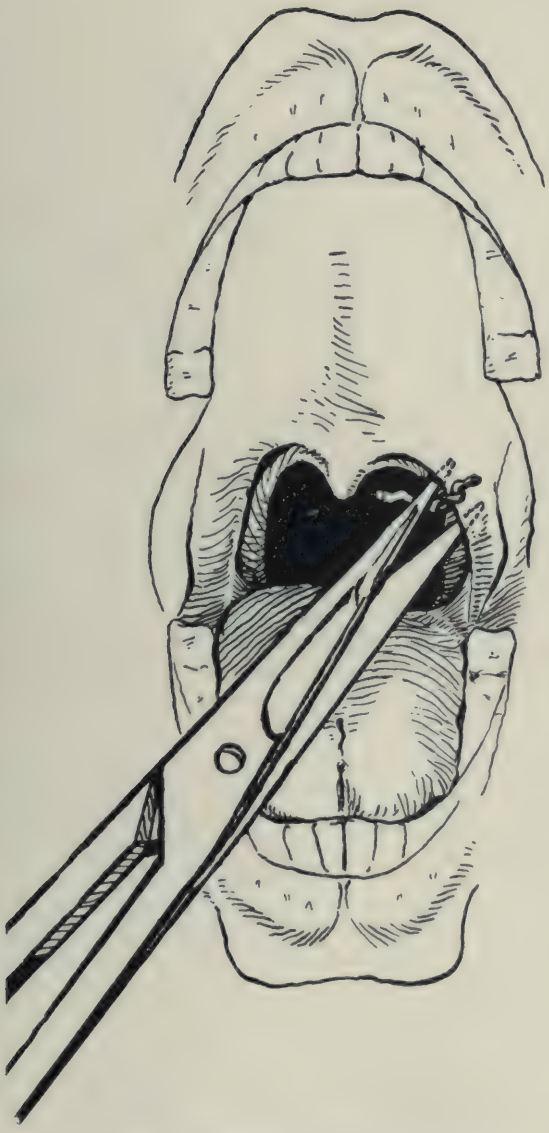


Fig. 17,—Removal of suture at the end of 24 hours.

gut, comes into view (Fig. 14). The suture hook, at the other end of the pillar pusher, pulls the thread forward while the eye itself slips back over the distal end of the silkworm, along the same path by which it entered. A double knot is tied (Figs. 15 and 16).

If the stitch has been taken in the right location, there will be no further bleeding. A second one is, however, occasionally required. By using the first as a palate retractor, the needle very readily passes through the approximated pillars in any location and to any depth that is desired. In one or two cases when the stitch has failed to catch the floor of the fossa, a ballooning of the pillars occurred with the formation of a hematoma underneath. This fact is mentioned to emphasize the importance of including in the suture the floor of the fossa with the pillars.

The suture is cut out at the end of twenty-four hours by straight blunt-pointed scissors introduced between the pillar margins. The closed scissors are then passed from the supratonsillar space down toward the base of the tonsil fossa, and the slightly agglutinated raw pillar surfaces, when thus separated, readily fall into their natural position (Fig. 17). Scarcely a mark can be seen to indicate the former location of the stitch. There is no edema, except when both sides have been sutured. With the exception of a slight sensation of tightness in his throat, the patient has been entirely comfortable while the suture was in situ.

DISCUSSION

Dr. J. E. Sawtell, Kansas City: Dr. Hayden has given a splendid demonstration of tonsil enucleation by the dissection method. In grasping the tonsil and making traction before cleavage, he speaks of drawing it forward instead of to the center. I prefer seizing the tonsil without any of its attachments and drawing it well to the center, as, by so doing, all its coverings are made taut and better access afforded for beginning cleavage. When the tonsil is thus retracted, its only coverings at the border of attachment, unless bound down by inflammatory adhesions, are the capsule and mucous membrane, with a small amount of cellular tissue intervening. In this way the mucous membrane can be divided and the capsule always reached at its first attachment to the tonsil. Hemorrhage is a subject in which most of us are interested. However, in this respect, it might be appropriate to group laryngologists into three classes: (1) Those who call themselves laryngologists, but never do any work. Their patients never have hemorrhage. (2) Those who hold a membership in the Ananias Club. By virtue of this their patients enjoy immunity from hemorrhage. (3) The class represented by the rest of us whose patients occasionally have hemorrhage. Dr Hayden's method for suturing the pillars is admirable. I believe, however, that the necessity for such a procedure is extremely rare. If we follow the method of torsion as taught by general surgeons long ago, all arterial hemorrhage can be safely controlled without either ligation or suturing the pillars.

In all my years of work I have depended on torsion alone. If we should be unfortunate enough to have a case with capillary oozing due to low clotting power of the blood, or some other constitutional disturbance, I could then see the advantage of suturing.

Dr. A. B. Middleton, Pontiac, Ill.: In support of Dr. Hayden's paper and method, I desire to say that the Eye and Ear Section of the Illinois State Medical Society, realizing this subject was of great importance, last year caused the speaker to send out a questionnaire to some 300 throat specialists, the most of whom are members of this society, with regard to the method of tonsillectomy they prefer and depend on, and 75 per cent. of the replies returned were emphatically in favor of the dissection and snare method.

Dr. Albert H. Andrews, Chicago: There are a number of things I would like to say about the comparative value of different methods, but I think by common consent we should let that subject alone. I noticed from the pictures that Dr. Hayden is using forceps such as I devised a good many years ago. The forceps as devised had rings that were complete on both parts of the handle and they had no snap. Some on suggested that one of the rings be opened so the wire loop would slip over. Many forceps are now made with an open ring, which is absolutely wrong. Any man who uses a wire loop large enough to slip over one ring does not understand the snare method. The loop should be made small, no larger than the tonsil, and the long diameter of the loop should be vertical instead of horizontal. It should be small enough to barely slip over the tonsil after the dissection is done. The further side of the loop should be slipped in between the tonsil and the posterior pillar and then the end of the cannula nosed in between the anterior pillar and the tonsil, and the whole tonsil will be removed. The tonsil forceps as now made have a snap on them. When the surgeon sees the snap, he is tempted to use it, and when the forceps take hold of the tonsil they begin to tear the tonsil, and it is very likely to tear out, while if only enough pressure is used to take hold of the tonsil it will not tear. I don't like an ether tube in the patient's mouth while I am working. Every breath blows ether into my face, and I don't like it. I prefer to have the patient well anesthetized, and then, in the majority of the cases, the surgeon should be able to remove both tonsils without having to give additional ether. The greatest hemorrhage comes from the removal of the tonsil, while the most difficult part is the dissection. If we dissect one and then the other, and remove the first and then the second, we get along far better.

Dr. H. G. Beaudoux, St. Paul: I think the consideration of suture is superfluous except in rare cases, as hemorrhages, primary or secondary, can usually be controlled either by hemostats or continued pressure. Secondary hemorrhages, occurring from a few hours after the operation to ten days, are usually easily controlled by the application of a sterile sponge in the tonsillar fossa. Personally, I have never seen any indications or necessity for ever using the suture to control a primary hemorrhage.

In a conversation which I had only a few weeks ago with Dr. Mathews, who does all his tonsillectomies under local anesthesia, and does little else all day long, he assured me that he had never seen an occasion for using a suture; and while I think he has more secondary hemorrhages proportionately than the average operator, it is not due to lack of sutures, but is perhaps due to the fact that he is not able to control his primary hemorrhages as well as he would if the patients were under a general anesthesia, and that he also is compelled to operate on many cases which would be considered bad risks. It is that class that swells his percentage

of secondaries, many of these occurring after the patients have returned home. They are cared for by local physicians without his being aware of their condition.

The important consideration about tonsillectomies is not so much the technic, the method, or the instruments used in operating. The important thing is to do good work; to do tonsillectomy and not pharyngectomy. Next to that, comes the care of the patient in regard to primary and secondary hemorrhage. We have found that if the primary hemorrhage has been unquestionably stopped before the patient leaves the operating room, then placed in bed on his abdomen, with the chest slightly elevated, face down, and a hypodermic of morphin given, though he may have had one before the operation, we have thus delayed the usual nausea and vomiting, which may be considered, in the majority of cases, the immediate cause for hemorrhages taking place within twenty-four hours after the operation.

Our experience during the past five years leads us to believe that this after care will do away almost entirely with secondary hemorrhages of any form.

Dr. C. F. Adams, Jersey City: The control of hemorrhage is the last stage of evolution in the removal of tonsils. I think the Academy is to be congratulated that we are not having calcium chlorid recommended as the first stage in preparing the patient for the operation, or watching how long it takes the blood to clot before we begin. The Jackson forceps to grasp the bleeding vessel, ligature thrown over that and tied, and the bleeding is stopped. That is simple and safe. It takes a little longer, but we can rely on it. There is sometimes necessity of a quick way of controlling hemorrhage, and that is at the base of the tonsillar fossa. The network of veins at this lower part is sometimes more difficult to isolate by a single ligature; here a deep suture is to be preferred. As for suturing above, it does not appeal to me. It is much more difficult than grasping the vessel, and it is not quite so reliable, in my opinion. The tying of the vessel is simplified by having the right hand in the mouth, the longer end of the thread outside, and running the forefinger along down the tonsil, and then drawing it up in that direction. (Ilustrating.)

Dr. Carmody, Denver: I would like to take exception to one point. The essayist says he uses warm ether. If the apparatus is correctly pictured, it is not warmed. You cannot warm ether volume. It is necessary to have a warming chamber in order to have warm ether vapor. I find I have very little retching and vomiting in using warm ether vapor. The vapor should have a temperature of 105 or 106 degrees when reaching the mouth.

Dr. A. A. Hayden, Chicago (closing discussion): The snare is of course worked down on the tonsil before it is tightened, just as Dr. Andrews suggests, although this point may not have been made clear in the paper. It seems most important that the finger rings on the tonsil-seizing forceps should be fenestrated; otherwise the application of the snare would be very difficult.

The author finds it necessary to stitch about one-fourth of his cases. It is very surprising to hear a man of Dr. Beaudoux' experience say that he never finds any such measure necessary. He also speaks of secondary hemorrhage occuring two hours after the operation. By the term secondary hemorrhage is universally meant the bleeding which follows the separation of the slough from the tonsil fossa. This of course does not occur before the seventh or the twelfth day.

A MODERN METHOD OF TONSILLECTOMY

G. E. Gwinn, M.D.
SAN ANTONIO, TEXAS

My only claim to originality in this method of tonsillectomy is my way of raising the encapsulated tonsil from its base (which rests on the superior constrictor muscle) sufficiently far to permit the vertical loop to be pressed outward beyond the greater diameter of the tonsil, so that, when the wire snare is released from the loop in the superior tonsillar fossa, it will follow in its circuit outward to the base of the tonsil, dividing it from its resting place and trimming the anterior and posterior pillars from the capsule in its downward and outward course.

In 1910, I read a paper on this operation before the Bexar County Medical Society at San Antonio, which was afterwards published in the *Jour. Am. Med. Assn.* under the title of "Simple Complete Tonsillectomy." Since that time my clinical experience has necessitated some changes, but the operation is performed practically the same way and with the same original instruments.

My attention was first called especially to this particular operation from the frequent examinations that I had made of different patients, who had undergone this operation and were not benefited, but even made worse. The cause for these conditions were various; the leaving of a part of the tonsil, cutting away one or both pillars, injuring the superior constrictor muscles, clipping the uvula, or doing all of the above mentioned. When performed with a knife or scissors, a history of hemorrhage, as a rule, followed the operation. Other unfavorable results were, dry throat from scar tissue, and inability to use the voice on account of the formation of scar tissue and injury having been done to the orificial muscles.

After a thorough study of the clinical anatomy of the tonsil and its anatomic relations, it was my judgment that this operation could be performed without sacrificing the normal structures, with great benefit to the patient, with the result of giving to the voice greater strength and volume, instead of weakening it.

After a careful examination of the many styles of instruments I had used myself, I found the cold wire snare preferable,

on account of less hemorrhage following its use. However, none of the instruments were made to conform to the natural formation of the throat; and, after some hesitation, I fashioned a snare and tonsil-seizing forceps in accordance with my own ideas, and these I found to meet every requirement.

I will not touch on pathologic conditions, but assuming that, after a careful examination, the tonsils are sufficiently diseased to be removed, the operation itself should be considered as a major surgical procedure. The patient should be sent to the hospital the night before, if possible, and the bowels thoroughly moved, a bath given, and no breakfast, if the operation is to be performed in the morning; and if at noon, or in the early afternoon, only a very light breakfast and no lunch. If an adult, I have given one-eighth gr. to one-sixth gr. morphin and one-one hundred and fiftieth gr. atropin one-half hour before the operation. If a child, I give only atropin, according to the age.

POSITION OF PATIENT

I prefer the recumbent position, and the head slightly lowered.

ANESTHETIC

I use the ether spray and think it is much better when it can be warmed while being administered.

CAUTION

In performing this operation, great care should be used so as not to injure the anterior pillars, which contain the palatopharyngeus muscles and from the anterior boundary of the sinus, or the posterior pillars which contain the glossopharyngeus muscles and form the posterior boundary of the sinus. These pillars meet above to unite with the soft palate and with the outer wall bounded by the superior constrictor muscle. The sinus tonsillaris is therefore a triangular depression in the lateral wall of the fauces, which partially envelops the faucial tonsil. Any injury to these muscles bounding the sinus tonsillaris lessens the action of the throat, has a deleterious effect on the voice, and prevents proper enunciation. As the tonsillar ascending pharyngeal and ascending palatine arteries supply these parts, it becomes more important that the technic should be such as not to injure the muscles bounding the sinus. Every good operator knows the importance of a strong light, so as to see clearly the field in which he is operating, as this

operation cannot be successfully performed when the field is obscure and touch is the only guide. I use an especially arranged globe, which throws a ball of light directly on the throat, and with the suction pump the field is kept clear at all times.

TECHNIC

After the patient is well under the anesthetic, I insert the mouth gag and test the reflexes by touching the back of the throat with my finger or applicator, and if I find the reflexes are not too active, I proceed as follows:

Standing on the right of the patient, with the assistants and instruments on the left, I turn the head to the right, so the light will show directly on the right tonsil, which I examine thoroughly to see if it is pendulous, deeply imbedded, friable, or fibrous, pillars adherent, or otherwise diseased. In making this examination, I first insert a soft rubber catheter through the nostril on the right and allowing the other end to come out through the mouth, and have the assistant pull it up. This raises the soft palate and shows clearly the anterior and posterior pillars. I also use pillar retractors to see if they are adhered. I then grasp the tonsil with specially designed tonsil-seizing forceps, one blade in the supratonsillar fossa and the other under the tonsil proper. I make sure that I have a very firm hold and have grasped only the tonsil. If the plica tonsillaris comes well up over the lower part of the tonsil, I endeavor to push it down and to grasp only the tonsil, lifting it completely out of its bed. I then slip the specially designed tonsillectomy snare over the handle of the forceps, and, by pushing the snare backward and outward against the angle of the jaw, at the same time holding the tonsil out of its bed and allowing the snare to pass between the anterior and posterior pillars, the tonsil will unfold, so to speak, directly through the aperture of the vertical loop. The wire being released first in the superior tonsillar fossa, the pressure of the wire is backward and outward, forming a half circle as it leaves the fenestrum, thus completely enucleating the tonsil, together with its investing capsule, without injury to the pillars or deeper muscles. If the plica tonsillaris has not been grasped with the lower blade of the tonsil-seizing forceps, it, too, will be left uninjured, as the wire in its circuit practically shells the tonsil out of its bed, leaving normal structures uninjured. The left tonsil is operated on in the same manner

as the right, after all bleeding has been stopped on the right side.

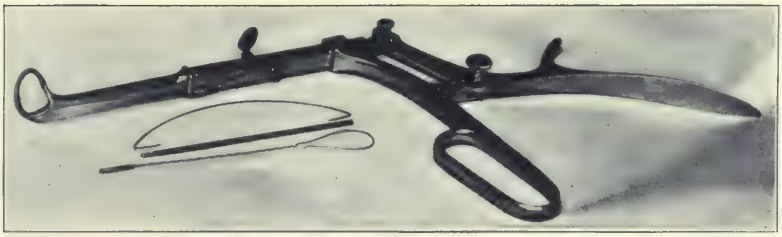
To my knowledge, no method of tonsillectomy without dissection had been performed prior to the reading of my first paper, which was published in the *Jour. Am. Med. Assn.*, 1911, lvi, 193-194. I have never had a case of alarming hemorrhage follow. I have had slight hemorrhages, which were easily controlled before the patient left the table. I never allow the patient to be moved till the throat is dry and free from hemorrhage. In controlling a slight hemorrhage, I have the nurse roll a small tight piece of gauze and run a thread through it and tie tightly, saturate it with compound tincture or benzoin, and press it well into the tonsillar bed, and hold it there for a few minutes, which readily controls all bleeding.

AFTER TREATMENT

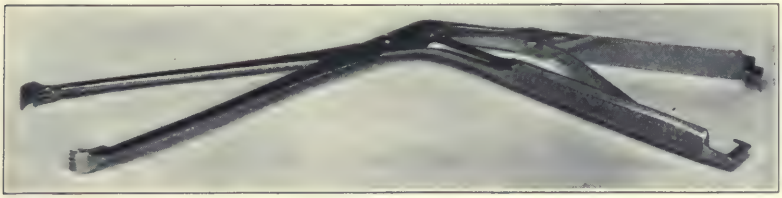
Ice pack is put around the throat and possibly scraped ice is used in the mouth, if the tendency to vomit is not too great. Patient remains in bed the rest of the day and night, with liquid diet for a couple or three days.

INSTRUMENTARIUM

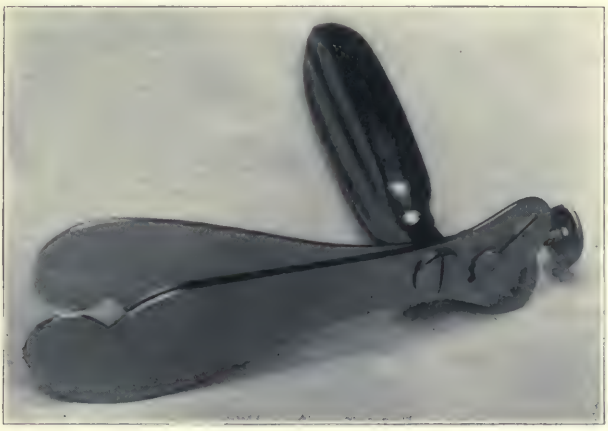
The construction of my first instrument was practically the same in general as this one, except the present one is made very strong, which is quite necessary, as the first one would not stand the heavy work and did not meet the requirements. The threading is different, although the instrument is made so the former way of threading may be used. There is a right and left loop arranged at an angle of about 45 degrees, which allows it to fit the side of the throat, either right or left, bringing the shaft across the tongue, which is thus depressed. There are three sizes of the loops, small, medium, and large. The medium and small sizes are about all that is necessary. Some like the large size in special cases. I use a No. 8 piano wire and have the loops made the same as in the Beck-Müller snare. The instrument is very strong and durable. Being hand-forged, it can be used without fear of breaking. The tonsil-seizing forceps are light and strong, making it easy to lift the tonsil from its bed without injury to structures. The saw teeth and fangs for catching and holding are patterned after a dog's mouth. I find these instruments have proved very satisfactory under trying circum-



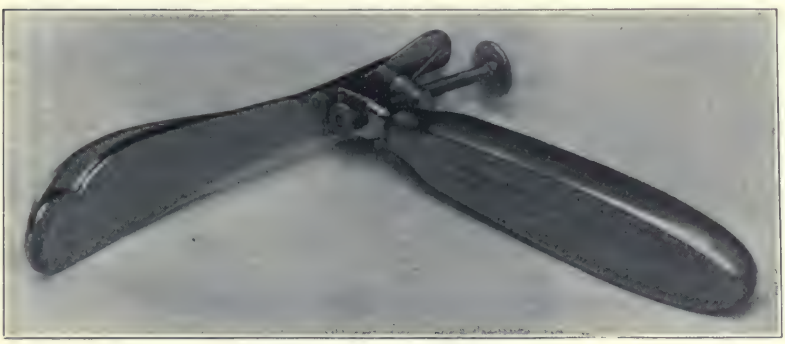
Vertical Snare.



Tonsil Seizing Forceps.



Adjustable Tongue Depressor (Open).



Tongue Depressor (Closed).

stances, and with tonsils that were submerged and very difficult to remove.

I have had an adjustable tongue depressor made which can be adjusted so as to fit any mouth, thus avoiding the trouble which every one has in holding down a broad, thick tongue with the ordinary depressor, which often slips to one side, or if pressed down in the center, the edges of the tongue will be forced up in the way and obscure the vision, necessitating a readjustment of the depressor. The rounding parts of the blades of this adjustable depressor are made so as to depress the tongue directly beneath the tonsils, and the notches on either side of the blades are for the purpose of self-retaining.

DISCUSSION

Dr. A. B. Middleton, Pontiac, Ill.: The main thing to do in removing a tonsil is to get it out without damaging the adjacent parts, by whatever method the operator feels is the best one indicated of those in his hands. It strikes me this instrument is a very good one in case the tonsil presents itself properly; but the tonsil that requires the greatest amount of skill is the one in which the parenchyma is practically gone, with but little more than the capsule remaining. It is almost impossible for one not experienced with an instrument of this kind to make a complete tonsil removal without first using some means of dissection. I am of the opinion that this instrument should not, any more than any other tonsil instrument be depended on in all cases of pathologically submerged tonsils; but it strikes me that it is a very valuable instrument in selected cases, and will save much time and trouble.

Dr. Joseph C. Beck, Chicago: The point here is not as to the method of removing tonsils, but the question of priority as to the method of removal of tonsils without preliminary dissection, and I speak here for Dr. Sluder as well as myself, in defense of that point. The American Laryngological, Rhinological and Otological Society has a committee on this question of priority, and the Academy should have one also. It gives me great pleasure to notice that the great inventions and ingenious things come often from the small cities. The men seem to have ability to do things, and I think Dr. Gwinn is to be encouraged and should be given credit for that work. In answer to the gentleman who opened the discussion, I would say, not only can we remove flat fibrous tonsils without any parenchyma, with capsule intact, but I have been working on another proposition and that is, to remove tonsils without the capsule. This may seem rather strange; but it will be an ideal method when the capsule is not taken out, and yet all of the tonsil is removed. I have done such a tonsil operation; then took the capsule out that remained, and found that there was no crypt left behind. The thing that I arise to discuss in this paper is the question of priority, and I think the doctor is perfectly right in presenting his subject from that point of view. I certainly do not see any resemblance to the Sluder method or my own, for I do not use the forceps to pull the tonsil into the ring.

Dr. W. W. Carter, New York City: In regard to technic, I wish to say that in all uncomplicated cases in children for the removal of tonsils, I used the Sluder method exclusively. But in those cases in which there has been a preceding peritonsillar inflammation, especially in adults, I have been accustomed to use the snare. I have not yet seen any grasping

forceps for the tonsil that did not have considerable breadth of grasp, and for that reason we are very apt to injure the pillar. Now, the most difficult cases that we have are the submerged tonsils. I devised a few years ago a little spiral tenaculum, which I have found useful. It consists of two spirals which compass half a circle each, and the tips of these tines are diametrically opposed, so that they can be inserted into the narrowest slit between the pillars. A half turn engages it and a half turn disengages it. Immediately after the tonsils are removed the patient's face is douched with ice water. The reflex action of this ice water on any hemorrhage is perfectly remarkable. I do on an average of thirty-six tonsillectomies a week in connection with my clinic at Gouverneur Hospital, New York, and these methods I have found satisfactory. In an experience of several thousand cases I have had no alarming hemorrhage.

Dr. J. M. Ingersoll, Cleveland: A word in regard to sponges for the control of hemorrhage. I have three sizes of gauze sponges made. They are rolled into a ball and caught with a thread so as to keep them round. Three sizes will cover every size of tonsillar fossa. To each sponge a piece of tape is sewed. A hemostat is attached to the tape by my assistant, and thus there is no possibility of swallowing the sponge.

A sponge of suitable size is placed in the fossa and held there with the finger long enough to allow the blood to clot. Then the sponge is carefully removed. This will control moderate hemorrhage. If the pressure for a few minutes does not control the bleeding, then the artery is tied just the same as would be done in any surgical procedure.

Dr. H. L. Pollock, Chicago: I desire to tell you in a few words how we control our tonsillar bleeding. After the tonsil is removed, a sponge of gauze, saturated with tincture of benzoin, is placed in the cavity, and the edges of the pillars are grasped with specially devised smooth-edged forceps. With an ordinary half curved needle, threaded with black silk, the pillars are sewn together, including a small portion of the gauze. This sponge is left in from twelve to twenty-four hours, usually twelve, and is then very easily removed. There is no danger of the patient swallowing the gauze, as the thread prevents this.

Dr. Bane: What is the after effect of a stitch through the pillars? Isn't there any injury done to the muscles in the pillars when it is swallowed?

Dr. Pollock: We have been doing this for two and one half years, and I have failed to see any damage from the stitch. We use the suction method of sucking all the mucus and blood from the throat, and there is seldom any retching.

Dr. Beck: It isn't put through the anterior pillar. It is put through the very edge, and is not tied so tight but that you can see the sponge in between. Dr. Bane's point is well taken.

Dr. G. Thomsen-von Colditz, Chicago: A method which I have found to be of great value in stopping hemorrhage following a tonsillectomy, is the use of horse serum. If after enucleation of the patient's tonsils, whether under general or local anesthetic, I find the bleeding does not stop on pressure, I inject 10 c.c. of horse serum into the patient's arm. In one case with profuse hemorrhage from both sides, about one hour after the operation, two clamps were applied; but the hemorrhage continued just the same. Five minutes after the injection of horse serum, however, the blood clotted and the bleeding stopped.

I have a coagulation test made in all adults, and in case the clotting requires more than ten minutes, I give 10 c.c of horse serum before operation. This lessens the bleeding considerably, and I have no post-operative hemorrhage.

Dr. G. C. Kneedler, Pittsburgh: There are but two types of hemorrhage that may give rose to trouble after tonsillectomy. The first

and most common is from the larger blood vessels, which should be taken care of with the hemostat, or if need by by ligation, before the patient leaves the operating room.

The second is from "bleeders," which may be obviated if a coagulation test be made previous to operating.

Dr. W. W. Carter, New York City: I think if we find after the operation is done that we have hemorrhage, the proper way is to stop it with a hemostat. I fail to see the use of suturing the pillars in all cases. The method would be impractical in dealing with the large number of cases that come under my observation in my clinic in New York. I don't believe I have sutured a pillar in three years, and I have not had a single case of alarming hemorrhage in a large number of cases.

Dr. Carmody, Denver: We take the coagulation test in every case, and know absolutely whether we are going to have bleeding or not. If we have a little oozing, washing the nasopharynx with boric acid solution will stop all oozing and get rid of the clots. I have used it for a number of years and have not had an infection of ears or accessory sinuses from it. If we have a bleeding vessel, we can stop it with forceps.

Dr. W. W. Carter, New York City: I think any method which is effective and practical is surgical. If, by vasomotor stimulation, we can stop capillary oozing a sufficient length of time for clots to form in the blood vessels, and we have no hemorrhage afterward, that is sufficient justification for the method.

Dr. J. C. Beck, Chicago: It is mostly from New York City that we get reports in the literature of lung abscesses and of pneumonia due to aspiration of blood, etc., during tonsil operations, and that is the reason why we adopted the sewing in of a sponge. I have seen cases in which the patients were returned to bed after a general anesthetic tonsil operation, and they aspirated blood. If you want to prevent all this, I think it is a good practice to sew in a sponge between the two pillars, especially in charity practice in large hospitals when the patients do not have a special nurse.

Dr. G. E. Gwinn, San Antonio, Tex.: I thank you for the generous discussion that you have given my paper. I do not dissect the tonsil, it makes no difference how deep and fibrous it is. I grasp the tonsil with my special tonsil-seizing forceps, raise it out of its bed, and take a periosteal elevator and push back the pillars and adhered membrane, also the capsule, if I can; and then, holding the tonsil firmly, I pass the loop of the snare over the handle of the tonsil forceps and back over the tonsil, which will invariably unfold itself through the loop.

Dr. Beck has just said he "leaves the capsule intact in the throat." I think that is a good thing to do, for I have noticed that there is not as much scar tissue after healing when it is left, but I am not always able to leave it.

In using the sponges I have long strings on them to hang out of the mouth, and grasp them with artery forceps.

I do not stitch the pillars over this sponge. So far I have had no hemorrhage, and what little capillary bleeding there was after the operation was easily controlled with the sponges. Should I have a hemorrhage, I should not hesitate to stitch the pillars over the sponge and leave it till the next day.

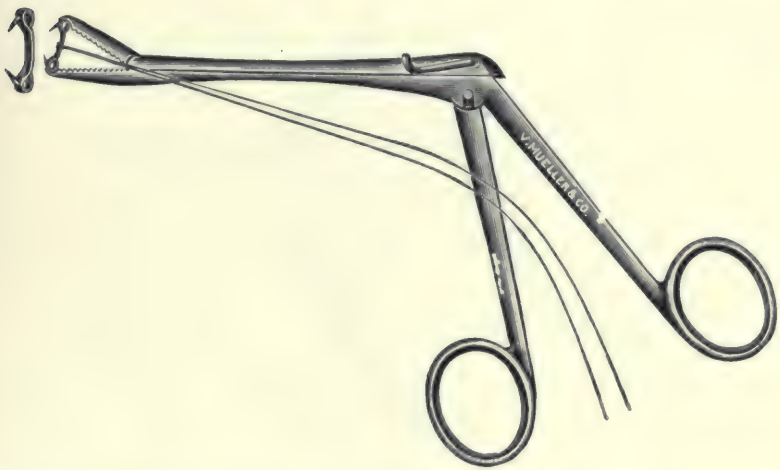
Now, as to grasping the tonsil, I have made the blades of the forceps sufficiently small to go between the pillars and grasp the smallest tonsil. The primary object is in getting a good, firm hold on the tonsil and retaining it. Seemingly the second hold is never so good as the first one. Douching with iee watcr I think unnecessary. I use the ice bag around the throat after the operation.

EXHIBITION OF INSTRUMENTS

NEW USE OF THE MICHEL CLIP

H. L. Pollock
CHICAGO

In removing the middle turbinate and the posterior end of the inferior turbinates, much difficulty is experienced by having the turbinate either slip away from the snare, or having it drop back and be lost in the nose, or be swallowed by the patient. To obviate this unpleasantness we have made use of a new procedure as first suggested by my associate Dr. Joseph C. Beck.

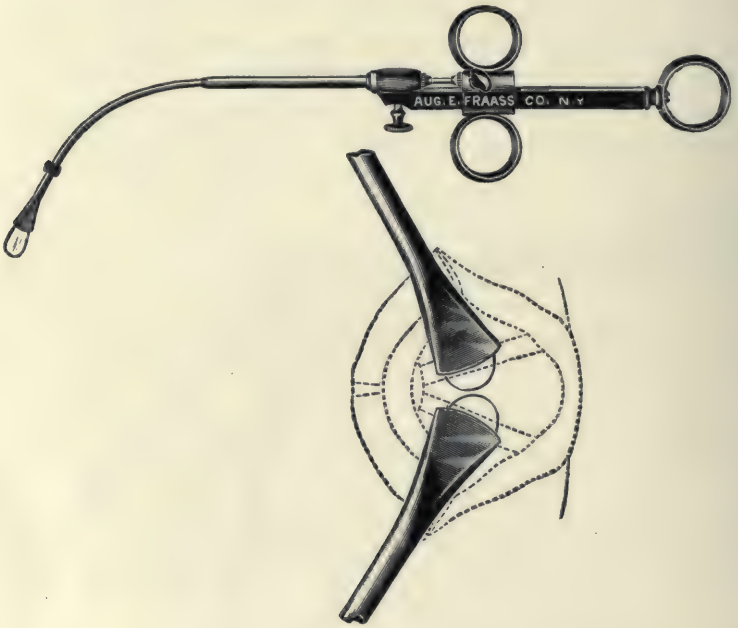


This consists of pressing into the end of the turbinate an ordinary Michel clip (Fig. 1), to which a long thread is attached. The clip is placed in position by means of alligator forceps. After grasping the turbinate with the clip, the turbinate is severed in the usual manner. The snare is pushed over the turbinate and the guiding thread is of great advantage in holding the turbinate in place, and also in preventing it from slipping down after it has been cut off. This procedure acts equally as well on hypertrophies of the posterior end of the inferior turbinate.

A NEW LARYNGEAL SNARE

T. S. Blakesley, M.D.
KANSAS CITY, MO.

It is necessary, at times, to consider a patient's objections and to operate on the larynx by the indirect method, which usually causes considerable difficulty in removing laryngeal growths on account of the sudden movements of the muscles of the larynx of the patient under cocain anesthesia, just at the moment an instrument is placed in position on the growth.



This new instrument consists of a snare on a Krause handle and is constructed with a soft, bendable cannula with a broad, flattened tip that carries the wire. This tip is bent at an angle to lie parallel to the left vocal cord, and with it the cord can be pushed to the side, if necessary, in order to reach growths lying below it.

These cannulas are made with a universal tip, so that it may be rotated in any direction.

One great advantage of this instrument lies in the fact that as soon as the growth is seen in the opening of the wire loop, the

instrument is closed instantly, while the flat tip is pressed outward, and the growth removed before the patient has an opportunity to move. I have had little difficulty in removing with it eight papillomas of the larynx.

It can also be used repeatedly without destroying the round loop, since, on account of its construction, the wire is not permitted to be drawn deeply into the flattened tip.

It can be procured from the August E. Fraass Co., New York.

A NEW PALATE ELEVATOR

T. S. Blakesley, M.D.
KANSAS CITY, MO.

This is a new palate elevator, of value in examination or treatment of the postnasal space, or in operations within the same.

It has all of the advantages of the Yankauer speculum with none of its disadvantages.

It consists of a cylindrically curved handle, offset from which is a side support for a tip, which is curved to fit the soft palate.

It is strong, light, easily cleaned, and not expensive.

With it the palate of even a refractory patient can be held by the operator, or an assistant, the angles of the instrument keeping the field clear for the operator.

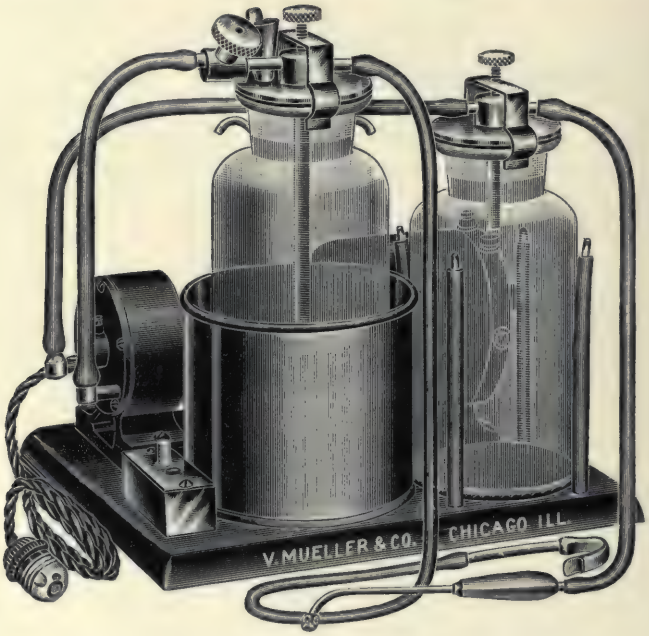
It permits direct examination of much of the postnasal space, and it has proved itself of distinct value; also when used reversed as a mouth searcher and tongue depressor.

It can be obtained from V. Mueller & Co., Chicago.

A NEW ETHER-SUCTION APPARATUS

SIDNEY ISRAEL, M.D.
HOUSTON, TEX.

This device is a combined suction and ether apparatus, the particular and distinctive features being that it is more portable than any other form of similar equipment presented up to this time; the exhaust and intake are utilized from one pump, the pump being attached to a single small motor which works on either the alternating or direct current. The exhaust air from the pump is attached to a bottle filled with ether that sits in a warming tank and from which we get the warm or hot ether vapor for anesthesia. From the intake side of the pump we get the suction, which is connected with a bottle the same size and is set along side of the ether bottle. Both bottles have control valves, so the flow of ether to the patient can be regulated or discontinued at the will of the anesthetist.

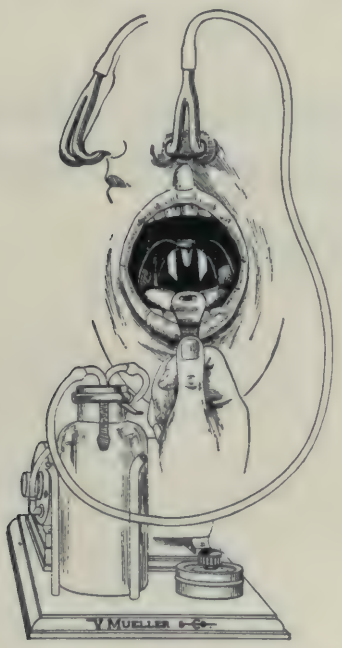


The apparatus occupies a small space and can be carried about with very little extra weight to one's equipment. That it works on either current with maximum efficiency is also a distinct advantage.

A NEW INSTRUMENT FOR ATTACHMENT TO SUCTION APPARATUS TO MAINTAIN A DRY FIELD IN TONSILLECTOMY UNDER GENERAL ANESTHESIA AND TO LESSEN OCCURRENCE OF POSTOPERATIVE PNEUMONIA

Sidney Israel, M.D.
HOUSTON, TEX.

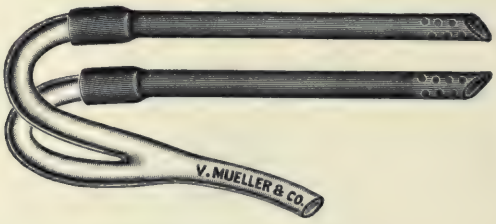
The aim of every surgeon is to operate with the field always as dry as possible, and to no one is such a condition of greater aid and importance than to the laryngologist. In the light of recent reports of far too great a number of cases of postoperative



pneumonia following tonsillectomy, when done under a general anesthetic, due possibly to the aspiration of blood, or highly infectious material from the tonsils, the writer was stimulated to look about for some means whereby this aspiration would be avoided, or reduced to such a minimum that the chances of this complication would be considerably lessened. The result has been ideal, and one feature that makes for its success and popularity is its simplicity in detail and efficiency in performance.

The device that I have the pleasure to submit has been thoroughly tried in numerous cases of tonsillectomy performed by the various methods in vogue today, namely, dissection, Sluder, Beck and LaForce, together with their popular modifications. It has also been used during suspension laryngoscopy and in several cases of surgery of the mouth, in each instance yielding the same gratifying success. Not only is the number of instruments introduced into the mouth lessened, but the added delay, assistance needed, and trauma by the repeated introduction of the suction tube are entirely dispensed with.

The means by which the oropharynx is rendered free from all blood, etc., and kept surgically dry during operation in the region is accomplished by the use of a metal tube that branches into the form of a "Y," and is curved on its long axis so it conforms approximately to the contour of the dorsum of the nose, in order



to lie flat and out of the way of the anesthetist and operator. To the ends of each of the metal tubes that form the prongs of the "Y" is attached a piece of rubber tubing, the size of which corresponds to a No. 19 French catheter, being about 5 inches in length, and each distal end has multiple small perforations over one-half inch at its extremity.

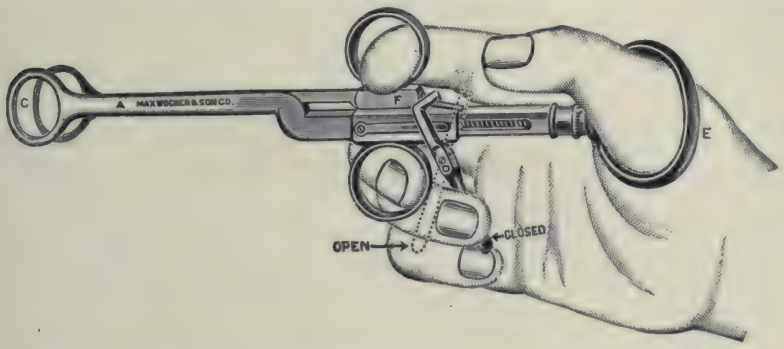
These tubes are inserted one into each nostril, along the floor of the nose and pass through the nasopharynx until they extend about one-half inch below and to each side of the uvula. To the stem, or single barrel, of the metal tube, from which the prongs of the "Y" spring, is attached the rubber tube that connects with any form of suction apparatus. Suction is then started and the contents of this region are rapidly emptied without further concern to the operator. Breathing is free, and the gurgling, bubbling sound that accompanies a general anesthetic in patients with excessive secretion of mucus, or, during operation, of escaping blood, is entirely eliminated, and the field kept dry throughout the entire operation.

THE TONSILLECTOME

A NEW TONSIL INSTRUMENT

FREDERICK W. LAMB, M.D.
CINCINNATI, OHIO

For several years I have been using Beck's tonsillectome in my tonsil work with the greatest satisfaction. I have, however, had difficulty at times with the wire loops. It is most annoying to have the wire slip out from under the set-screw, or slip through the hole in the stylet, when it should be closing down behind the tonsil. To obviate this difficulty, I have devised a blade to take the place of the wire. The cutting edge is dull and rounded, so



that in effect it is much the same as a wire. It is held in place by a set-screw, which passes entirely through a hole in the blade. Therefore, it cannot slip. It is in reality a permanent, non-breakable, "nonslippable" snare which never requires rethreading. I have used this instrument in a great many cases and find that it enucleates thoroughly and completely the tonsil in its capsule, leaving a smooth, clean fossa, and with a minimum amount of hemorrhage.

INDEX

PAGE

Abscesses, Dental Accommodative Asthenopia 196
Addresses—
  President 1
  Vice President 207
Antrum, Mastoid, Early Drainage of 275
Artery, Central, Occlusion of 39
Articulation, Temporomandibular, Surgery of 245
Astenopia, Accommodative 196
Bone, Transplants of, Rhinological Surgery in 286
Campimeter 94
Carcinoma, Primary, Middle Ear 300
Cartilage, Transplants of, Rhinological Surgery in 286
Cataract, Treatment of 188
Chalazion, Causing Changes in Corneal Curvature 169
Choked Disk, Renal 25
Choroid Disease, Ring Scotoma in 13
Clip, Michel, New Use of 361
Congenital, Defect of Retinal Pigment Layer of Iris 151
Conjunctivitis—
  Parinaud's 113
  Tularensis 135
Cornea, Changes in Curvature 169
Deaf, Problems of the 257
Defect, Congenital, Retinal Pigment Layer of Iris 151
Dental Abscess, Accommodative Asthenopia in 196
Diphtheria, Acute Otitis Media, Cause of 270
Drainage
  Early, in Mastoiditis 275
  Through Mastoid Antrums in Radical Mastoid Operation 275
  Early, Safeguard in Mastoid Operation 275
Ear, Middle Primary Carcinoma 300
Elevator, New Palate 363
Epithelioma, Limbus of 118
Ether-Suction, New Apparatus 364
Fields, Visual, in Pellagra 85
Glands, Internal Secretion of 220
Glaucoma—
  Iridotasis in 108
  Simplex 137
Hemorrhage—
  Nasopharyngeal, Control of 227
  Nature and Control 227
Hyphophysis, Secretion in Ear, Nose and Throat Affections 211
Internal, Secretory System of Eye 5
Internal Secretions 211
  Relation to Otolaryngology 220
Interpretation of Stereo-Roentgenograms 239
Iridotasis, in Glaucoma 108
Kitten Eyes, for Operative Technique 177
Lid, Tumors 169

Limbus, Epithelioma of 118
Limitation, Tonometer of 121
Mastoid Operation, Early Drainage in 275
Mastoid, Stereo-Roentgenograms in 239
Michel Clip, New Use of 361
Middle Ear, Primary Carcinoma 300
Muscles, Inferior Oblique 172
Superior Rectus 172
Nasopharynx, Hemorrhage in, Control of 227
National Military Home, Cataract in 188
Nephritis, Acute, Caused by Sinusitis 267
Oblique, Inferior, Overaction of 172
Occlusion, Superior Branches of Central Artery 39
Operation, Tonsil Technic in 331
Optic Nerve, Ring Scotoma in Disease of 13
Osteomyelitis, of Skull 315
Otitis Media, Acute 270
Otolaryngologist, Enlargement of His Field 207
Palate, New Elevator 263
Paresis, Superior Rectus 172
Parinaud, Conjunctivitis 113
Pellagra, Visual Fields 85
Perimeter 94
Plague, Squirrel 135
Poliomyelitis, Tonsils Atrium of Infection 324
Problems of the Deaf 257
Renal Choked Disk 25
Retina, Detachment, Scleral Trephining in 156
Retinal, Pigment Layer of Iris 151
Roentgenograms, Stereo 239
Scotoma, Ring, Relation to Choroidal and Optic Nerve Disease 13
Secretory System 5
Sinusitis, Frontal, Cause of Nephritis 267
Skull, Osteomyelitis of 315
Snare, Laryngeal, New 362
Squirrel Plague 135
Suction Apparatus, New, in Tonsillectomy 365
Surgery, Temporomandibular Articulation 245
Therapeutics of Tuberculin 75
Tarsus, Excision of 100
Technic, Operative 177
Tension, in Glaucoma 137
Tonometer 121
Tonsils, Atrium of Infection in Poliomyelitis 324
Tonsil, Operative Technic 331
Tonsillectome, New 367
Tonsillectomy, Modern Technic 353
Trachoma 100
Transplants, Bone and Cartilage 286
Trephining, Scleral 156
Tuberculin, in Uveitis 75
Tuberculosis, Retinal Vessels 60
Tumors, Lid 169
Vein, Central, Thrombosis 39
Vessels, Retinal, Tuberculosis of 60

RE
1
A44
v.21
Biological
& Medical
Serials

American Academy of
Ophthalmology and
Otolaryngology
Transactions

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY

STORAGE